The effect of forest management options on forest resilience to pathogens

Roberts, Michaela; Gilligan, Chris; Kleczkowski, Adam; Hanley, Nicholas; Whalley, Elizabeth; Healey, John

Frontiers in Forests and Global Change

Published: 10/02/2020

Peer reviewed version

Frontiers in Forests and Global Change

Section: Forest Disturbance

The effect of forest management options on forest resilience to pathogens

Roberts, M.[1,2,3*], Gilligan, C.A.[4], Kleczkowski, A.[5,6], Hanley N.[7,2], Whalley, A.E.[8] & Healey, J.R.[3]

[1] Social, Economic and Geographical Sciences Group, James Hutton Institute, Aberdeen, UK

[2] Department of Geography and Sustainable Development, University of St Andrews, St Andrews, UK

[3] School of Natural Sciences, Bangor University, Bangor, UK

[4] Department of Plant Sciences, University of Cambridge, Cambridge, UK

[5] Department of Mathematics and Statistics, University of Strathclyde, Glasgow, UK

[6] Faculty of Natural Sciences, University of Stirling, Glasgow, UK

[7] Institute of Biodiversity, Animal Health and Comparative Medicine, University Of Glasgow, Glasgow, UK

[8] Warwick Business School, University of Warwick, Coventry, UK

***Correspondence:**
Michaela Roberts
Michaela.roberts@hutton.ac.uk

Keywords: Tree disease, epidemiology, forestry, pathogen, silviculture, forest management, invasive species

Abstract

Invasive pathogens threaten the ability of forests globally to produce a range of valuable ecosystem services over time. However, the ability to detect such pathogen invasions – and thus to produce appropriate and timely management responses – is relatively low. We argue that a promising approach is to plan and manage forests in a way that increases their resilience to invasive pathogens not yet present or ubiquitous in the forest. This paper is based on a systematic search and critical review of empirical evidence of the effect of a wide range of forest management options on the primary and secondary infection rates of forest pathogens, and on subsequent forest recovery. Our goals are to inform forest management decision making to increase forest resilience, and to identify the most important evidence gaps for future research. The management options for which there is the strongest evidence that they increase forest resilience to pathogens are: reduced forest connectivity, removal or treatment of inoculum sources such as cut stumps, reduced tree density, removal of diseased trees and increased tree species diversity. In all cases the effect of these options on infection dynamics differs greatly amongst tree and pathogen species and between forest environments. However, the lack of consistent effects of silvicultural systems or of thinning, pruning or coppicing treatments is notable. There is also a lack of evidence of how the effects of treatments are influenced by the scale at which they are applied, e.g. the mixture of tree species. An overall conclusion is that forest managers often need to trade-off increased resilience to tree pathogens against other benefits obtained from forests.

1 Introduction

Invasive species present significant threats to natural and planted forests (Wingfield et al., 2015; Leibhold et al., 2017; Muzika, 2017), and can, in combination with climate change, create 'mega disturbances' which disrupt forests worldwide (Millar and Stephenson, 2015), leading to large ecological, economic and social losses (Hill et al., 2019). While invasive species research often focuses on animal and plant invasions, forest ecosystems are also threatened by invasive microbial pathogens. Pathogens have the potential to disrupt timber and non-timber benefits provided by forests, and the need for a coordinated effort to tackle such invasive species is being increasingly recognised (Wingfield et al., 2015). In this context, to be considered invasive a pathogen does not need to be non-native to the region, but rather through an increase in its abundance produce a widespread negative impact on a given ecosystem (Warren, 2007; Carey et al., 2012)).

Managing invasive pathogens presents unique challenges not associated with controlling invasive plants and animals, including insects. The cryptic nature of infection by pathogens, particularly at the beginning of their life cycles, means that many invasions remain undetected until trees become symptomatic, by which time the pathogen is often already widespread (Liebhold et al., 2017; Muzika, 2017). Even after infection has been detected, identification of the causal pathogen is normally reliant on examination of spores and/or DNA sequencing. As a result, many pathogens have remained unidentified or misidentified, restricting our capacity to manage invasions effectively (Wingfield et al., 2015; Wingfield et al., 2017). Genetic variation amongst microbial pathogens is greater than even that found between the plant and animal kingdoms, and as such these pathogens have highly varied life histories. This not only creates challenges in designing control measures for pathogens as a general invasive threat, but also in tackling individual pathogens, which can present unfamiliar life history traits (Wingfield et al., 2017). These complex and often still unknown life histories, as well as their potential for relatively rapid evolution, restrict our ability to predict which pathogens will become invasive, and how any invasion will progress (Ghelardini et al., 2017).

Tree pathogens threaten the ability of forests to deliver ecosystem services over the long-term. The importance of phytosanitary measures, such as quarantine, to reduce the risk of invasive tree pathogens reaching a country or a given forest have long been recognised (Wingfield et al. 2015). However, as rotation lengths from establishment to harvest of a forest tree crop generally last for several decades, the high rate of arrival of new pathogen species to locations around the world means that any newly established crop may potentially be subject to many new pathogens before it reaches maturity. This poses a particular challenge for forest managers. So far, management responses have largely been restricted to reactive measures taken after the presence of a given pathogen has been detected, by which time economic and ecological damage costs can rarely be avoided. This restriction on the ability of forest managers to respond until a specific pathogen has been detected raises the important question of whether and how forests should be planned and managed to maximise their resilience to the threat of future unknown pathogens.

To help address this challenge, a recent extension of epidemiological modelling ("epi-economic modelling") has linked the economics of forest management practices to the impacts of tree pathogens across a range of primary and secondary infection rates and damage costs (Macpherson et al., 2017a; Macpherson et al., 2017b; Macpherson et al., 2018). This approach is based on an epidemiological paradigm (Kleczkowski et al., 2019), whereby the population of trees is divided between healthy and susceptible individuals, and infected

individuals. Mathematical models capturing the infection process can take different forms but the simplest equation is

$$\frac{dI}{dt} = \left(r_p + r_s I(t)\right) S(t) \qquad (1)$$

with $S(t)$ denoting the number of still healthy but susceptible trees, r_p and r_s the rate of primary and secondary infection, respectively, *I(t)* currently infected trees within the unit, and d*I*/d*t* the rate of appearance of new infections (Brassett and Gilligan, 1988).

The fundamental difference between primary and secondary infection is epidemiological. Primary infection is the invasion of the population of trees within the forest management or landscape-patch unit from an external source, e.g. an infected population of trees in another unit, and requires management at the boundary or beyond. Primary infections can also occur from a reservoir of inoculum in alternative hosts, or in soil or dead plant material when sites are replanted. Thus, the source is 'external' to the population under threat albeit occupying the same parcel of land. Secondary infection is transmission from currently infected trees within the unit's population to its susceptible trees, driven by multiplication, dispersal and infection of inoculum. Hence management activity in that forest unit can influence secondary infection and reduce epidemic spread. The rates of primary and secondary infections in equation (1) capture the whole range of factors, including the susceptibility of individual trees to infection as well as the dispersal characteristics of the pathogen.

Fundamental to this paper is the recognition that resilience of a forest is linked to its response to invasions by forest pathogens. This response, in turn, is influenced by the management practices aimed at the prevention of such invasions, their control and, if control is unsuccessful, the mitigation of their effects. The modelling framework described above and its extensions, have successfully been used in describing spread and control of tree pathogens in forests (Macpherson et al., 2017a; Macpherson et al., 2018). However, application of such an approach to inform forest managers about how to increase forest resilience against future pathogen threats requires empirical evidence about the effects of forest management options on tree pathogen primary and secondary infection rates, and rates of forest recovery.

The key question in ecological and economic applications of the concept of resilience is "resilience of what, to what?" (Walker et al., 2010; Matsushita et al., 2018). The focus of this study is forests that are managed predominantly for timber production and in this case the most relevant concept of resilience is the one termed "engineering resilience", which comprises two main components: "resistance" to the initial impact of a disturbance agent (in this case the invasion of a tree pathogen) and "recovery" towards the previous state or functioning of the forest ecosystem (Pimm, 1984; Holling, 1996; Grimm and Wissel, 1997; Newton and Cantarello, 2015). In the literature reviewed in our study, the measured response variables that give the best indication of "resistance" (at the scale of the forest ecosystem or stand) were level of individual tree infection or mortality, and the best indicators of "recovery" were rates of natural regeneration or stand-level growth rates of all surviving or subsequently established trees. We assume throughout the paper that managers are concerned with a single spatially contiguous unit of forest, because this is the scale at which most studies are carried out. However, the size of this unit may be highly variable, and we focus only on net increases or decreases in resilience. The majority of studies available for our review assumed that provisioning of timber was the ecosystem service of greatest importance resulting from the state or functioning of the forest.

The impacts of forest management on tree pathogens have been the subject of many recent reviews. Each has tended to focus on a single pathogen, such as white pine blister rust (*Cronartium ribicola*) (Schoettle and Sniezko, 2007; Hunt et al., 2010; Ostry et al., 2010; Zeglen et al., 2010), *Phytophthora ramorum* (Valachovic et al., 2010), ash dieback (*Hymenoscyphus fraxineus*) (Pautasso et al., 2013) or dothistroma needle blight (*Dothistroma septosporum* and *D. pini*) (Bulman et al., 2016). As a result, the findings have been highly variable, with recommendations largely dependent on the pathogen considered. Alternatively, pathogens have been considered alongside insect pests (Waring and O'Hara, 2005; Liebhold et al., 2017; Muzika, 2017) or other general threats to forest ecosystems (Jactel et al., 2009; Jactel et al., 2017). Reviews focused on broader forest resistance to the threat of tree pathogens have been limited to considering only the effect of tree species mixtures on the spread of and damage caused by pathogens (Pautasso et al., 2005; Prospero and Cleary, 2017) or of more general principles of interactions and ecosystems services (Boyd et al., 2014). Overall, the local context of the forest and pathogen have been recognised as important in directing management responses.

The objective of the current study is to synthesise the evidence for the effect of forest management options on forest resilience to tree pathogens. The scope is broad, including all of the main categories of forest management variables and all tree pathogen species. However, animal pests, invasive plants and abiotic threats such as fire were excluded. A second objective is to forge an explicit link between forest resilience, forest design or silvicultural management practices and epi-economic modelling grounded in plant and tree epidemiology (Macpherson et al., 2017a; Macpherson et al., 2017b; Macpherson et al., 2018). Thus, we seek to assess the empirical evidence for the effect of each forest management option on the three key elements of primary infection, secondary infection and subsequent forest recovery, in order to inform forest management decision making to increase forest resilience, and to identify the most important evidence gaps to motivate future research.

2 Methods

We carried out a literature review using a systematic search method to identify published sources of empirical data on the relationship between forest management and resilience to tree pathogens. We conducted an initial search of the peer-reviewed literature through Web of Science, using search strings to identify papers on all of forest management, pests, pathogens or disease, and resilience, excluding medical papers and those concerned with food supply, using the Boolean search string of:

> TS = ((((*forest* OR wood* OR tree*) AND (manage*)) OR silvicult*) AND (pest* OR disease* OR pathogen*) AND (exposure OR resist* OR recover* OR spread OR risk OR suscept* OR transmit* OR dispers* OR infect*) NOT (medicin* OR clinic* OR pharma* OR foodborne OR food-borne OR mycorrhizal OR biomedic* OR mosquito OR tick OR lyme* OR malaria*)) NOT SO=(medicin* OR clinic* OR pharma* OR biomedic*) NOT WC=(medicin* OR clinic* OR pharma* OR biomedic*)

This search (run on 27/06/2017) returned 3534 papers. The papers were screened first by title, then abstract, and finally full text to identify papers reporting original empirical data on the effects of forest management on tree diseases caused by pests or pathogens, which retained 599 papers. We then excluded papers that only covered tree pests (362 papers), were

only concerned with tropical forests (85 papers) or orchards (235 papers), were concerned with tree breeding (74 papers), or were entirely review (23 papers) or theoretical modelling (21 paper) studies (note papers may be present in more than one category). Removal of tree pests also included removing papers concerning insect vectors, because in the majority of cases the distinction between direct damage and vectoring of a pathogen could not be determined. This procedure retained 81 papers. As many forest management actions are not reported in the published literature, searches were also run in TREESEARCH, the research portal for the US Forest Service, using the following search string:

> (disease OR fung* OR pathogen OR bacteri* OR oomycete OR virus) AND (((tree OR forest* OR wood) AND manage*) OR silvicult*)

and the UK Forestry Commission website, using the search string:

> disease fung* pathogen bacteria* oomycete virus

Restrictions on the search terms for each search engine prevented identical searches from being carried out. The search in TREESEARCH returned 158 documents, of which 12 were identified as relevant and containing data. The Forestry Commission website returned 58 documents, of which three were identified as relevant and containing data. Together with literature identified from the reference lists of retained papers and identified reviews (nine papers), and further search terms added to account for fertilizer application (four papers), the final reference list contained 114 papers and reports. This list was further refined to include only papers whose reliability, robustness and applicability to forest management could be assured. Studies which were purely descriptive, lab-based or considered only pathogen presence, rather than impact, were excluded. The remaining 109 papers included within the review were scored for strength of evidence based on whether they were correlative or experimental, and whether single or multiple sites had been considered, within single or multiple forests.

We organised papers by management technique, treating each technique reported within a single paper independently, and then by pathogen type. We have included a broad range of management options, including forest design planning, site preparation for forest establishment, tree species diversity, silvicultural system and individual silvicultural actions. These categories were not pre-determined but were decided through reading the papers.

To assess the outcome of each management technique we classified the results of each study as strong positive or strong negative (relationship observed in all sites within the study), weak positive or weak negative (relationship observed in at least one site within the study, with no sites showing the opposite relationship), no relationship, or mixed (both positive and negative relationships observed across sites). A technique was therefore considered to have an overall positive or negative impact where multiple studies, or a robust single study across multiple forests, found the same result, and there were no robust studies reporting a contradictory result. If studies reported contradicting results, we considered the outcome of this technique to be mixed unless the results were weighted heavily in one direction, and the contradicting study was considered to be of low robustness. Where only a limited number of studies was available this was identified as a weakness in our conclusions. A fuller description of this critical appraisal of the studies is provided in the Supplementary Material, together with the full outputs of the search and critical appraisal in the table ‘All_studies_classification’.

The large variation in tree species, pathogens, and management techniques considered, as well as limited reporting of the particulars of management, prevented us from conducting a formal meta-analysis.

3 General trends

Our review of the literature revealed a clear bias towards a small number of highly damaging pathogens. Studies into *Armillaria* and *Phytophthora* species comprised 35% of all studies identified (14% and 21% respectively), and we found no studies on the effects of forest management on bacteria or viruses. Studies mainly covered commercially valuable host tree species, with equal coverage of conifers and broadleaves (Table 1).

A geographical bias was also evident, with 57% of studies based in North America, and California and Oregon coastal forests alone accounting for 23%. The majority of the remaining papers originate from Europe (23%), with eight papers from Oceania and two from Asia. Studies reporting only from natural tropical forests had previously been excluded. Only a single paper (Cleary et al., 2013) reported results from more than one region (Table 1).

The response variables most commonly reported were mortality, disease incidence and disease severity. The only indicator of forest recovery (defined in the Introduction) that was widely reported was of subsequent tree growth rate, though this was often only measured over the short-term. There was minimal reporting of rates of tree natural regeneration. Studies generally reported outcomes in terms of symptoms of forest disease, and few papers considered the mechanisms connecting forest management to these outcomes. The distribution of studies amongst each forest management variable and each response variable is summarised in Table 2.

4 Identification and management of sources of primary infection

As explained above, the concept of primary infection (cf. Eq. (1)) captures the pathways by which the pathogen enters the forest unit of interest. These primary infections can occur from other forest units, for example by wind or water movement of inoculum, from alternative hosts, by movement on machinery and other human-mediated activities, or by transmission from soil inoculum.

4.1 Connectivity

The importance of connectivity for the conservation of forest ecosystems at a landscape scale is well recognised (Lindenmayer et al., 2006). However, connectivity can increase the risk of transmission of infection from one forest patch or unit to another, including from outside the region of interest. For tree pathogens connectivity does not just refer to spatial proximity, but also any connection through which inoculum may spread to a forest unit, such as via streams, wind, fog, animal (e.g. insects, fur, feathers) or human vectors (e.g. vehicles, recreation). The scale at which connectivity is important also varies with dispersal mechanism, and can be large in the case of flying animal and vehicle vectors. These ‘least cost’ (resistance), but not

necessarily shortest-distance, pathways have been shown to be important in models accounting for the spread of pathogens (Ellis et al., 2010).

The impacts of connectivity on tree diseases have predominantly been studied in coastal forests of California and Oregon (USA). Total forest area within a landscape, correlated with connectivity, predicted increases in incidence (Meentemeyer et al., 2008a) and severity of *P. ramorum* in *Notholithocarpus densiflorus* (tanoak) (Haas 2011) and *Umbellularia californica* (California bay laurel) (Condeso, 2007; Meentemeyer et al., 2008a; Meentemeyer et al., 2008b; Haas, 2011). Disease was considered only in these species because they represent the most prevalent hosts for *P. ramorum* within this landscape. *Phytophthora ramorum* incidence increased closer to streams in one site, suggesting that streams are also an important connectivity pathway for dispersal of this pathogen, though this relationship appears to depend on site topography and fog movement (Peterson et al., 2014). Connectivity through human vectors was related to higher concentrations of *P. ramorum* being isolated in soil from sites surrounded by larger human populations (Cushman and Meentemeyer, 2008), and increased mortality of *Chamaecyparis lawsoniana* (Port Orford cedar) caused by *Phytophthora lateralis* was found in sites intersected by a road (Jules et al., 2002). *Phytophthora lateralis* has been isolated from water used to wash vehicles and boots, providing further evidence of the importance of human vectors to the spread of this pathogen (Goheen et al., 2012). Within California and Oregon coastal forests, black stain root disease (*Leptographium wageneri*) is also concentrated at the roadside (Hessburg, 2001).

Forest connectivity via spatial proximity (Condeso, 2007; Meentemeyer et al., 2008a; Ellis et al., 2010; Haas, 2011), streams (Peterson et al., 2014; Havdova, 2017), or roads (Hessburg, 2001; Jules et al., 2002; Goheen et al., 2012) shows a consistent positive relationship with disease incidence and severity compared with less well connected forests. However, the limited geographical range of these studies and their predominant focus on pathogen spread through soil restricts their applicability to other systems. There is also little mention of the effect of land cover in the matrix between forest patches that could affect pathogen dispersal. When addressing the increased risk of pathogen infection due to high connectivity, forest managers must also balance the extensive benefits that connected forests can have for biodiversity and some ecosystem services against increased vulnerability to disease (Lindenmayer et al., 2006). Future studies should identify and quantify pathogen transmission along different types of pathway connecting forest units and via different vectors in order to assess risks. This should help selection of management strategies to reduce the risk of primary infection of forest units.

4.2 Previous land use

Many tree pathogens, in particular root rots, can persist in soils following tree felling. Siting new plantations on previously forested sites may therefore increase the risk of infection due to high inoculum load in the soil. Here the soil acts as an ‘external’ reservoir of inoculum for primary infection to initiate an epidemic in a newly planted tree population. However, research into the effects of previous land use is limited, due to the relative rarity of studies into forests established on previously non-forest land.

Naturally occurring *U. californica* trees on former grassland sites within *Quercus* forests in northern California had lower incidence of *P. ramorum* than *U. californica* growing in long-term forest areas (Meentemeyer et al., 2008a). However, this relationship was not reflected in

Italian *Abies alba* (silver fir) plantations, where *Heterobasidion annosum* infection rate was significantly higher in forests established on former pastureland. Although *A. alba* planted on former pastureland was expected to be exposed to lower inoculum load, these trees were less healthy due to exposure to adverse environmental conditions resulting from previous intensive land use, potentially increasing susceptibility to disease (Puddu et al., 2003).

While previous land use could be expected to affect forest resilience to tree diseases, research on this is rare, and the findings amongst published studies are not consistent. This is likely to be due to the large variation in previous land use types, and particulars of previous land management, amongst the studies. However, such research is likely to increase in relevance for rotational forest systems, where the previous species planted in the unit may be considered. In some countries, including the UK, new forests are also being planted on land not forested in recent history in order to increase carbon capture in response to climate change, and natural tree regeneration is occurring due to abandonment of agricultural land, (Poyatos et al., 2002).

4.3 Site preparation

Site preparation methods can either introduce pathogens into an area where they were not previously found or reduce forest resistance to primary infection and hence increase the initiation of local disease spread. Previously felled sites contain remnant stumps, root fragments and brash, which may be a source of primary infection through spread of pathogens over time (from a previous tree population to a new one). Nonetheless, stumps resulting from thinning or partial cutting can also act as a source of secondary infection within the current tree population. Although this coarse woody debris is important for forest biodiversity more generally (Hartley, 2002), it acts as a reservoir for many pests and pathogens, increasing exposure of newly established trees to inoculum. Stumps can also provide a nutrient source for inoculum of pathogens with saprotrophic activity. In response to this risk, site preparation may include stump removal or chemical treatment, in some cases accompanied by removal of part of the root system through raking. The origin of stumps can also be important for identifying the risks posed, with clearcut stumps having lower infection rates than stumps resulting from thinning (Bendz-Hellgren and Stenlid, 1998). Burning presents an alternative option to remove woody debris or reduce pathogen survival, but has other risks and environmental costs, such as to habitat quality or native fauna, or it can in fact increase disease incidence in the case of *Rhizina undulata* (Wingfield and Swart, 1994). In some cases site preparation also includes application of fertilizer, which may reduce impacts of pathogens on tree health through increasing tree nutrient concentrations, especially of calcium and magnesium (Anglberger and Halmschlager, 2003; Halmschlager and Katzensteiner, 2017). However, fertilizer application (especially of nitrogen and phosphorus) that increases tree growth rate can lead to nutrient inbalances that increase susceptibility to pathogens (Jactel et al. 2009).

Root rots have the highest potential for management through stump treatment and have unsurprisingly been the subject of the greatest number of studies. Methods of stump treatment may be physical or chemical. Infection of forest stands by *H. annosum* has long been known to be increased by any tree felling resulting in cut stumps that are susceptible to colonization from air-borne basidiospores (Woodward et al., 1998). The most extensive study of stump removal incorporated five sites from Canada and Scandinavia subject to infection by the conifer root rot pathogens *H. annosum* sensu lato (*s.l.*), *Armillaria ostoyae* and

laminated root rot (*Phellinus sulphurascens*). The severity of infection and its contribution to mortality were monitored in stands that had been subject to removal of stumps, either as part of the whole tree or in a separate operation following felling, with raking to remove larger roots occurring in one site, compared with controls where no stump removal took place (Cleary et al., 2013). Stump removal was clearly associated with reduced disease incidence and tree mortality up to 21-50 years after treatment. One of these sites was then studied in more detail by Morrison et al. (2014), who confirmed that over 40 years after treatment, stump removal had reduced the rate of mortality in the next rotation of trees by 14% across all species. Notably, for *Pseudotsuga menziesii* (Douglas fir), *Pinus contorta* var. *latifolia* (lodgepole pine), *Larix occidentalis* (western larch), *Thuja plicata* (western red cedar) and *Picea engelmannii* (Engelmann spruce), stump removal reduced the mortality rate due to *A. ostoyae* and completely eliminated the occurrence of mortality in *P. menziesii* due to *P. sulphurascens.* These findings support those of a previous study of *P. menziesii* further south in Washington State and Oregon, where mortality due to *P. sulphurascens* was significantly reduced by pre-planting stump removal (Thies and Westlind, 2005).

There has been a long history of incidence of *H. annosum* in the plantations of *Pinus sylvestris* (Scots pine) and *Pinus nigra* ssp. *laricio* (Corsican pine) in Thetford Forest, UK. A series of experiments showed that colonization of stumps by air-borne basidiospores of *H. annosum* can be greatly reduced by stump treatment with spores of *Phlebiopsis gigantea*, a non-pathogenic basidiomycete. However, a series of long-term experiments have shown that only stump removal achieves adequate reduction in mortality into the second plantation rotation (Gibbs, 2002). The importance of removing stumps was further supported in studies of *P. sylvestris* infected by *H. annosum* in Sweden, where infection rate was higher in trees closer to infected stumps (Swedjemark and Stenlid, 1993). In contrast, in New Zealand *Pinus radiata* plantations infected by *Armillaria novae-zelandiae*, while inoculum load in stumps was high, this did not lead to greater infection within the forest after 19 years compared with forests where stumps were removed (Hood et al., 2002; Hood and Kimberley, 2009).

In addition to retained stumps, root fragments from felled trees can act as reservoirs of pathogen inoculum. Few studies have reported beneficial effects of root removal, although Shaw et al. (2012) found that greater intensity or thoroughness of removal of roots that acted as a source of *A. ostoyae* inoculum did significantly reduce the incidence of infection and resultant mortality in *Pinus ponderosa* after 35 years, though the study concluded that its benefits are unlikely to exceed its costs. In contrast, several studies have reported increases in infection following root removal. Negative impacts of mechanical root removal treatments can occur through increasing the dispersal of pathogen inoculum. Root raking has been found to move infected root fragments closer to the soil surface, although this was not associated with increased infection rates (Morrison et al., 1988; Morrison et al., 2014). The incidence of western gall rust was found to be positively associated with sites that had undergone a variety of methods of mechanical site preparation to disrupt slash, forest floor and mineral soil layers, compared with control sites (Roach et al., 2015). Similar results were observed in *Castanea dentata* (American chestnut) infected by chestnut blight (*Cryphonectria parasitica*). In a study of plantations on reclaimed mine land in Ohio, USA, the site preparation treatments of deep ripping to 1 m depth resulted in a higher incidence of chestnut blight cankers on seedlings than in those plots ploughed and disked to 30 cm depth, though this still exceeded the incidence of cankers for trees in control plots (Bauman et al., 2014).

Prescribed or natural fire reduces pre-planting inoculum load through either burning of stumps, root fragments and woody debris, or through killing of the pathogen due to high

temperatures. However, results from burning are not consistent. Naturally occurring fires in Californian coastal redwood and mixed-evergreen forests led to reduced isolation rates of *P. ramorum* in symptomatic trees for one and two years following fire, however incidence of the pathogen increased by the second year, associated with the number of surviving symptomatic *U. californica* trees which acted as an inoculum reservoir (Beh et al., 2012). A prescribed burn treatment in a *Pinus palustris* (longleaf pine) forest in South Carolina, USA, was associated with increased mortality through *H. annosum* after 8-10 years, linked to reductions in tree health caused by the fire (Cram, 2010). Varied burning regimes in conifer forests in Ontario, Canada, had no impact on *Armillaria* sp. root rot (Whitney and Irwin, 2005).

Fertilizer application has mixed impacts on disease severity. Increased damage by twisting rust fungus (*Melampsora pinitorqua*) was found on *Pinus pinaster* (maritime pine) that had been fertilized with phosphorus compared with no fertilizer controls (Desprez-Loustau et al., 2016). However fertilization of *Picea abies* (Norway spruce) did show a reduction in severity of infection by *Sirococcus* shoot blight (*Sirococcus conigenus*), linked to improved tree health (Anglberger and Halmschlager, 2003; Halmschlager and Katzensteiner, 2017). In *P. menziesii* seedlings fertilization with potassium and nitrogen had no impact on mortality due tolaminated root rot (*Phellinus weirii*) (Thies et al., 2006), and potassium, nitrogen and sulphur fertilizers had not impact on *Armillaria* spp. root disease in mixed conifer forests in Oregon (Filip et al., 2002).

Overall, removal or treatment of tree stumps as a source or receptor of pathogen inoculum has a positive effect on forest resilience to tree disease, through reduced infection of trees retained on the site or newly planted trees. However, studies are concentrated on root rots. Stumps, and other dead wood material, are also known to be important in survival of populations of a number of invertebrate forest pests, and there is an evidence gap about their significance as a source of inoculum of a wider range of pathogens with airborne spores that infect the shoots of trees. We found that studies of root fragment removal and burning give more mixed results and are under-researched. A future research priority is to assess the trade-offs between reducing inoculum levels using such treatments and the damage they cause to retained trees (e.g. through wounding), which can increase their susceptibility to infection.

5 Management of sources of secondary infection

Secondary infection (cf. eq. (1)) refers to transmission of a pathogen between trees within a region of interest (forest unit). Secondary infection therefore captures the direct transmission component of epidemics that depends upon the number of currently infected individuals. Although this typically relates to an outbreak situation between trees of a similar age, of particular concern for forest management is secondary infection from mature trees to seedlings, often planted to form the crop in the next forest rotation. Actions that increase environmental stress on a tree, thus reducing its vigor, are also likely to increase the rate of secondary infection.

5.1 Tree species mixture and diversity

Effects of tree species mixture, i.e. planting two or more species rather than a monoculture, or increasing tree species diversity, i.e. through the number of species planted together or as an

indirect result of other silvicultural actions, on forest resilience to tree diseases have been extensively studied, with good coverage of both tree and pathogen species in sites across Europe and North America. Tree species diversity effects have been the subject of recent review papers. These recognise that greater diversity is associated with decreased tree mortality caused by pests and pathogens, identifying reduced access to hosts and increased distance between hosts as potential mechanisms for reducing secondary transmission as an epidemic progresses (Pautasso et al., 2005; Bauhus et al., 2017; Jactel et al., 2017; Prospero and Cleary, 2017), as well as potentially providing habitat for more species that deliver natural biocontrol (Bauhus et al. 2017). However, such results may be context-specific (Heybroek, 1982), and depend on whether the invading pathogen is a host specialist or generalist. We have included within this section observational studies of variation in pathogen incidence with tree species diversity as well as experimental studies with species mixtures. Their coverage is summarised in more detail in Tables 2 and 3. Provenance and breeding of trees for resistance is also an important aspect of forest resilience to tree pathogens, but was outside the scope of this review.

There is general agreement across studies that increases in tree species diversity are associated with an increase in forest resilience with respect to invasive pathogens. In sites across Europe, more diverse forests were associated with lower levels of disease incidence (Nguyen et al., 2017). These findings are supported by experimental units of broadleaf trees in Germany, where fungal infections of the most susceptible tree species were reduced as species diversity increased (Hantsch et al., 2014a). This pattern was also recorded in studies in North America, where higher tree diversity was associated with lower occurrence of *Fusarium* sp. canker in *Acer saccharum* (sugar maple) (Bergdahl et al., 2002), and higher tree species diversity was linked to a lower incidence of *P. ramorum* in Californian oak forests (Haas, 2011; Haas et al., 2016). Stands with higher tree diversity were also found to have lower mortality rates of *P. menziesii* caused by *P. ramourm* (Ramage et al., 2012). In an experiment in Germany, planted with individual tree-scale species combinations in 5 x 5 m plots, high tree species diversity in the same plot reduced the amounts of fungal infection in trees of *Quercus petraea* (sessile oak) (Hantsch et al., 2014a). Only a single study contradicted these results, showing in Spain and Italy that *Armillaria* spp. presence and abundance in pure *A. alba* stands was lower than in mixed-species stands (Oliva et al., 2009).

Diversity amongst clones in monocultures can also affect resilience to tree disease. Willow rust (*Melampsora epitea)* causes decline in many short rotation *Salix* spp. (willow) crops. In an experiment in Northern Ireland, UK, McCracken and Dawson (1998) found that increasing the diversity of clones reduced mortality due to rust in the most susceptible clones only, but no further improvements in survival were seen when moving from 5- to 20-clone mixtures. Mixed-clone sites were also found to have later onset of disease during the first four years of growth, but showed no difference after this time (McCracken and Dawson, 1997). However, following observations of a subsequent experiment over two three-year harvesting cycles, Begley et al. (2009) found no consistent effects of mixtures on reducing *M. epitea* on the most susceptible *Salix* genotype. They concluded that any benefit of mixture planting will be dependent on there being sufficient genetic diversity between the genotypes.

An important mechanism cited for the benefit of species mixture is the dilution of trees of species susceptible to a given pathogen by individuals of non-host species. However, experimental results of this effect are variable, and the specific composition of species in a mixture is found to be important. In an experimental study in Minnesota with seedlings of six conifer and four hardwood tree species planted in three mixtures differing in the proportion

of individual tree species, the relative proportion of susceptible conifers or resistant broadleaves had a significant effect on mortality associated with *Armillaria* sp. root infection (Gerlach, Reich et al. 1997). These findings were supported by longer-term experiments in British Columbia, in which *P. contorta* var. *latifolia* had lower mortality, due predominantly to *A. ostoyae*, when grown in mixture with *Betula papyrifera* (paper birch) and *T. plicata* both of which have low susceptibility to this pathogen (Morrison et al., 2014). However, no benefit was found when growing it in mixture with the highly susceptible host species *P. menziesii*. In contrast, for 30-year-old *P. menziesii* no reduction in root pathogen-caused mortality rate was found from growing it in mixture with non-host species. Also in British Columbia, experimental removal of naturally regenerated broadleaves *B. papyrifera* and *Populus tremuloides* (quaking aspen) generally caused a 1.5- to 4-fold increase in mortality of planted *P. menziesii* for 3–5 years due to *A. ostoyae* infection, though the effect depended on removal method (Gerlach et al., 1997; Baleshta et al., 2005; Simard et al., 2005). In Southwest Lapland and in North Karelia, Finland, modelling of observational data showed that the incidence of *M. pinitorqua* on young *P. sylvestris* was greater in the presence of both *Populus tremula* (aspen, also a host species), and *Salix* spp. (willows, not known to be host species but said to be indicators of higher soil moisture and fertility) (Mattila et al., 2001; Mattila, 2002).

There is variation amongst conifer species in their susceptibility to *H. annosum* butt rot, and in southern Sweden the presence of less susceptible *P. sylvestris* was found to decrease the incidence of this pathogen in trees of the more susceptible *P. abies* (Linden and Vollbrecht, 2002). This effect increased notably up to a relative abundance of 50% of *P. sylvestris* but not above that proportion. Experimental plantings in Germany showed that, overall, tree species diversity in mixtures of 30 mono-specific 8 x 8 m sub-plots reduced the level of foliar pathogen infestation of susceptible *Quercus* spp. by *Erysiphe alphitoides* and *E. hypophylla* (powdery mildew) at the plot level (Hantsch, 2013). The presence of the highly susceptible *Quercus* spp. increased the plot-level pathogen load but resistant species such as *P. abies* decreased it.

Variation in the impacts of tree diversity on resistance to tree pathogens is likely related to tree species identity in the same forest unit (on a scale from individual adjacent trees up to ca. 50 m), with tree characteristics beyond simply host or non-host being important. Indeed, this may also be the main driver of any detected effects of species diversity within forest stands. Severity of infection of *Fraxinus* spp. to the pathogen *H. fraxineus* was highest in the presence of *Quercus robur* (pedunculate oak) and lowest in the presence of *Acer* spp. and *Abies* spp., both non-hosts. However, this result from an observational study of forests across the Czech Republic does not prove causation by the tree species themselves, but may be linked to variation in site environments (Havdova, 2017). In the rigorously-designed planted experiment studied by Hantsch et al. (2013), for trees of *Tilia cordata* (small-leaved lime) both fungal species richness and infestation level (predominantly of *Passalora microsora* and *Asteromella tiliae*) were reduced by tree diversity in the plot. For non-host tree species the effect of their proportion in the plot on infestation level in *T. cordata* varied from significantly positive (for *Fagus sylvatica*, European beech) to negative (for *P. sylvestris* and in some years for *P. abies*). Similarly, infestation of the leaves of *Q. petraea* increased with the proportion of *Fraxinus excelsior* (European ash) in the plot and decreased with *P. abies*. In a rehabilitated bauxite mine site in Western Australia, experimental mixed planting with non-host species reduced mortality of the susceptible host tree species *Banksia grandis* caused by soil inoculation with the pathogen *Phytophthora cinnamomi* only when grown in mixture with *Acacia pulchella*, but not with four other non-host *Acacia* species (D'Souza et

al., 2004). The mechanism was suggested to be that mixture with *A. pulchella* reduced the soil inoculum level. These results provide strong evidence of the importance of tree species-identity effects.

In general, higher tree diversity improves forest resilience to tree diseases. However, a major mechanism in this effect has been found to be linked to the identity of the tree species present (i.e. species composition). Highly susceptible species show the largest reductions in pathogen presence and impact with increases in tree diversity. There is also evidence that greater benefit for such susceptible tree species can be obtained if they are mixed with trees not susceptible to the pathogen. It is important that future experiments are designed in a way that allows separation of effects due to species identity from those due to species diversity *per se* through careful consideration of species and mixtures tested. There would also be benefit in future studies determining the role of alternative mechanisms causing species mixture or diversity effects on pathogen infection, such as relative levels of inter- and intra-specific competition and their impacts on tree condition. While the resilience of mixed-species forests to individual host-specific tree pathogens may increase at the whole-forest ecosystem scale, the sum of components of these ecosystems are vulnerable to a larger number of tree pathogens than is the case for single-species forests. More work needs to be done to understand how the dynamics of different classes of pathogen affect the trade-off associated with the epidemiological risks and benefits of mixed cropping at different scales. Mixed-species forests generally increase the costs and complexity of management. Therefore, in reaching evidence-based forest management decisions, biological and ecological considerations need to be combined with economic analysis that explicitly considers the multiple costs and benefits over time of alternative management responses to invasive pathogens.

5.2 Tree establishment under different silvicultural systems

For many tree species, planting or natural regeneration under shelterwood leads to better establishment than in open conditions (e.g. after clearcutting) (Raymond and Bédard, 2017). This form of silviculture can also be beneficial for plant (Hannerz and Hanell, 1997) and bird (King and DeGraaf, 2000) biodiversity. Increased vigor of trees grown in shelterwood sites may increase resistance to tree disease, however the retained canopy trees may also act as a source of inoculum.

Across studies, the relative incidence of pathogen infection between shelterwood and clearcut sites shows high variation even within sites, and between tree and pathogen species. In study locations across the USA, *Pinus strobus* (eastern white pine) experienced either increase (Ostry, 2000), decrease (Katovich et al., 2004) or no change (Katovich et al., 2004) in incidence of *C. ribicola* when grown in shelterwood compared with clearcut sites. Across the same locations, *Armillaria* sp. root rot of *P. strobus* was less common in shelterwood than in clearcut sites (Ostry, 2000). A history of clearcutting increased the incidence of *Neonectria ditissima* in trees of *Betula* spp. (birch) (Ward et al., 2010).

Amongst selection or retention systems, the size of felling gap has been suggested to influence forest resilience to tree diseases, but it is acknowledged that this varies with tree and pathogen species. The most rigorous experimental study was carried out in a pine forest in Minnesota (USA), in which *Pinus resinosa*, *P. strobus* and *P. banksiana* (red, eastern white and jack pine) were planted within felling gaps of 0.3 ha and 0.1 ha, within plots with

evenly-spaced retained overstorey trees or in an unfelled control. Incidence of shoot blight (predominantly *Diplodia pinea*) in dead seedlings was significantly less in the 0.3 ha gap treatment than in the control for both *P. strobus* and *P. banksiana*, but not for *P. resinosa*. In contrast, *Armillaria solidipes* incidence was significantly greater in one or both of the two gap sizes than the control for all three tree species. Gall rust (likely caused by *Cronartium quercuum* f. sp. *banksianae*) was observed only in *P. banksiana*, and its incidence was significantly greater in the small gaps than the control (Ostry et al., 2012). In no cases were any significant differences in pathogen incidence found amongst the two gap sizes and the evenly-spaced retention systems. Similar variability in response to silvicultural treatments was seen in an experimental study of *Pinus radiata* (Monterey pine) forest infected by *Fusarium circinatum* (a causal organism of pitch canker) in California. Here, incidence of the disease was greater in seedlings growing in intermediate-size (0.10 ha) gaps than in smaller (0.05 ha) or larger (0.20 ha) gaps (Ferchaw et al., 2013). However, gap size was not found to affect the odds of seedling survival. Tree position with respect to a long-term forest edge has been found to influence crown dieback caused by the pathogen *H. fraxineus* in retained trees of *F. excelsior* in an observational study following a tree harvest in Estonia (Rosenvald et al., 2015). The level of dieback and mortality resulting from *H. fraxineus* was less for trees adjacent to the pre-existing forest edge than those in the centre of the harvest gap. Such effects of tree position need to be considered in studies of other pathogens and locations.

Understanding of the impacts of the range of alternative silvicultural systems on forest resilience to tree diseases is poor, with relatively few studies. It is not surprising that the available evidence shows little consistency across pathogen and tree species, given the wide variation in their transmission pathways and modes of infection. Transmission to seedlings of pathogens that spread via root contact is expected to be greater in shelterwood or other even-retention or small-gap selection systems. This effect may be less so for pathogens that disperse via airborne or water dispersal. Similarly, amongst trees, light-demanding species that show greatest vigor in open clearcut or large gap sites are likely to be less susceptible to infection in such site conditions. In contrast, more shade-tolerant species may be less susceptible in shelterwood or small gap systems where they are less vulnerable to environmental stress. However, such deductions, and in particular the net effects of any trade-offs between the effect of site conditions on the rate of pathogen infection and on the level of seedling vigor, need further empirical research. It can be expected that the net outcome will vary amongst tree and pathogen species.

Individual silvicultural systems differ from each other in several different component silvicultural operations and resulting stand conditions, which are addressed in turn in the sections below.

5.3 Canopy cover

Differences in forest canopy cover at different stages of the forest growth cycle is one of the obvious distinctions amongst different silvicultural systems. It is also influenced by decisions over specific silvicultural operations, e.g. tree species selection, planting density and thinning. Canopy cover affects microclimate, solar irradiation and air flow, all of which can alter the survival and dispersal of pathogens within a forest. Although it could not be distinguished as a separate effect in the reviewed literature, canopy cover would also be expected to affect movement of animal vectors of disease. We found only three studies explicitly investigating the impacts of canopy cover and their results conformed to the

627 expectation for the different types of pathogen species, given that greater canopy cover is
628 associated with higher air humidity, but lower sub-canopy wind speeds. Two studies in
629 California mixed evergreen forest found a positive relationship between canopy cover and
630 severity of infection by *P. ramorum*, a species whose dispersal and colonization is dependent
631 on high humidity (Condeso, 2007; Ellis et al., 2010). In contrast, in British Columbia, *C.*
632 *ribicola*, a species whose spores can disperse successfully through dry air, was reduced in
633 sites with higher canopy cover (Campbell, 2000). As the effects of canopy cover clearly
634 differ so much between different species it is not possible to draw conclusions across tree
635 pathogens in general. In order to provide a stronger evidence base for the relative effect of
636 different silvicultural systems in limiting the rate of secondary infection of tree pathogens,
637 new research into the mechanisms by which canopy cover alters pathogen dispersal and
638 infection is a high priority.

639

640 **5.4 Tree density**

641 High tree density reduces the distance between potential host individuals and would therefore
642 be expected to increase rates of pathogen spread by secondary infection within a forest. This
643 effect is likely to vary among pathogen species, with a greater effect seen for pathogens that
644 spread via root contact than for those with only airborne dispersal. Dispersal via animal
645 vectors is also likely to be affected by tree density, though this could not be distinguished as a
646 separate effect in the reviewed literature. Variation in total tree density can result from many
647 causes, e.g. initial density of planting or natural regeneration or reduction in density due to
648 intensity of thinning or other forms of selective felling. Reduction in density of individual
649 host species can occur as a result of mixture with other species (reviewed in section 5.1).
650 Studies that reported on the effects of thinning as an operation are reviewed in the following
651 section (5.5).

652 We found only one study testing the relationship between tree density and the incidence of a
653 pathogen species that spreads through root contact. In Minnesota, USA, broadleaf and conifer
654 seedlings were planted in several species mixtures in recently logged sites at four different
655 densities, ranging from 0.25 to 2 m spacing. In this study the effect of closer spacing on
656 mortality was not significant (Gerlach et al., 1997). Airborne pathogens have been subject to
657 much more extensive study. The intensity of *P. ramorum* infection increased in an
658 observational study of mixed evergreen stands in California with higher densities of the three
659 primary host species (Dillon et al., 2014). This positive relationship between tree density and
660 pathogen incidence or impact has been observed for a range of other tree and airborne
661 pathogen species and locations, including crown dieback of *F. excelsior* due to *H. fraxineus*
662 in forests across the Czech Republic (Havdova, 2017), mortality of *P. sylvestris* due to snow
663 blight (*Phacidium infestans*) in Sweden (Burdon et al., 1992), and infection level by *M.*
664 *pinitorqua* of both *P. sylvestris* in Southwest Lapland and in Northern Karelia, Finland
665 (Mattila et al. 2001, Mattila, 2002), and *P. pinaster* in France (DesprezLoustau and Wagner,
666 1997). However a number of other studies find no relationship between pathogen incidence
667 and tree density (McCracken and Dawson, 1998; Bishaw et al., 2003; Piirto and Valkonen,
668 2005).

669 High tree densities increase susceptibility to a broad range of tree pathogens, both those
670 spread by root contact and airborne spores, although this effect is not universal, with many
671 studies showing no relationship. It is likely that the relationship between tree density and
672 pathogen prevalence is not linear but characterised by thresholds at both low and high

673 densities. For most pathogen species forests with a high load are unlikely to see changes in
674 pathogen spread through reduction in tree density, as the probability of secondary infection is
675 likely to remain high even with relatively large distances between trees. Similarly, once
676 distance between trees exceeds the normal dispersal distance of a pathogen, further increases
677 in distance would be expected to have a smaller effect. We found no clear evidence of effects
678 of forest structure per se, though many studies did report on the progression of disease during
679 the development of planted stands. Priorities for future applied research would be to improve
680 understanding of the mechanisms of tree density effects and identify thresholds in tree
681 density related to pathogen load. In considering the role of tree density as a factor in species
682 diversity effects on susceptibility to pathogens, an important source of evidence from future
683 research would be to distinguish the influence of absolute tree density from that of the
684 relative density of individual species and from the effect of forest structure (e.g. tree size
685 heterogeneity). Thus, research should specifically compare the effects of reducing host
686 species density by increased spacing in monoculture versus dilution by planting in mixture
687 with non-host species.

688

689 **5.5 Thinning**

690 Thinning may be carried out as a planned action to increase production of the highest value
691 timber from a forest, to improve other components of stand condition, or in response to
692 damaging disturbance events, including tree pathogen outbreaks. In the latter case, thinning
693 can take the form of salvage cutting, where dead or dying trees are removed, or sanitation
694 cutting, which targets trees highly susceptible to disease, with the intention of reducing forest
695 inoculum load. The latter type of thinning to remove susceptible trees will be considered in
696 the next section (5.6). Thinning to improve growth or other components of tree vigor, through
697 reduction in tree density (section 5.4), could also be expected to improve resilience to tree
698 diseases. However, studies show a large variation in forest response to thinning actions.
699 Negative impacts could be attributed to the resulting stumps, whose cut surfaces are
700 susceptible to infection (compare section 4.3), wounding of remaining trees, or due to
701 increased traffic within managed areas, increasing pathogen spread by vectors (Jules et al.,
702 2002; Cushman and Meentemeyer, 2008; Goheen et al., 2012).

703 *Armillaria* sp. root rots have been the best studied pathogen with regards to thinning impacts,
704 with studies consistently finding evidence that thinning increases pathogen infection. Weights
705 of *Armillaria* sp. isolated from the soil increased with past thinning intensity in *A. alba* stands
706 in the Spanish Pyrenees (Oliva et al., 2009) and incidence of *A. ostoyae* infection was higher
707 in experimental units that had been thinned for a range of conifer species in British
708 Columbia, compared with paired unthinned stands (Morrison et al., 2001). This result was
709 also observed in experimental *P. menziesii* plantations in Oregon (Rosso and Hansen, 1998)
710 and Idaho (Entry, 1991), as well as for *A. luteobubalina* infection of *Eucalyptus diversicolor*
711 (karri) in Western Australia, with no increased growth rate of trees retained after thinning
712 (Robinson, 2003). In *P. radiata* plantations in New Zealand thinning also increased stand-
713 level infection of retained trees by *A. novae-zelandiae* partly through infection from stumps,
714 however the incidence of infection diminished as the stumps decomposed, leaving no effect
715 of thinning after six years (Hood, Kimberley et al. 2002; Hood and Kimberley 2009).

716 Studies of other species of root rot also predominantly show an increase in infection with
717 thinning. In 15-year old *P. menziesii* stands in northern California, incidence of *L. wageneri*
718 was much higher in thinned than in unthinned stands (Harrington et al., 1983). This finding

719 was confirmed in a much more extensive survey of *P. menziesii* plantations in southwest
720 Oregon, which found that incidence of *L. wageneri* was significantly higher in thinned than
721 unthinned stands, though this effect was not apparent in all the studied forests (Hessburg,
722 2001). In an experimental study of *P. abies* stands in Sweden, the probability of stump
723 infection was much higher following thinning in the summer than the winter (Thor and
724 Stenlid, 2005). This pattern persisted following a second thinning of these plots (Oliva et al.,
725 2010). However, infection rates following summer thinning were greatly reduced by a range
726 of chemical and biological (spores of *P. gigantea*) treatments of the cut stumps (Thor and
727 Stenlid, 2005), and plots with stumps treated with urea had much lower overall mortality
728 (Oliva et al., 2008). Only a single study recorded a decrease in infection following thinning,
729 with reduced mortality of *P. ponderosa* due to *L. wageneri* ten years after experimental
730 thinning in north-eastern California (Otrosina et al., 2007). Thinning may also be
731 accompanied by measures to remove root fragments, particularly where thinning was carried
732 out with the intention of tackling root rots. However intensive root removal can be associated
733 with wounding the roots of retained trees, which increased the risk of infection of *P.*
734 *tremuloides* by *Armillaria* spp. (Pankuch et al., 2003).

735 Thinning has highly variable effects on tree diseases besides root rots. The most frequently
736 studied pathogens infecting tree shoots have been dothistroma needle blight of *Pinus* spp.
737 caused by *D. septosporum* and *D. pini*. A comprehensive review of management and control
738 of these pathogens was provided by Bulman et al. (2016). They found that in Australia, Chile,
739 New Zealand and USA, reducing stand density by thinning reduced disease levels. However,
740 ongoing experiments in the generally wetter climates of Great Britain and British Columbia
741 have not shown a notable effect on disease incidence. No benefits were found of thinning for
742 control of these pathogens, or *Lecanosticta acicula*, in *P. radiata* plantations in northern
743 Spain (de Urbina et al., 2017). Thinning was reported to reduce damage of *P. contorta* var.
744 *latifolia* due to *E. harknessii* across 27 plantations in southeastern British Columbia (Roach et
745 al., 2015). However, no significant effect of thinning of *P. contorta* var. *latifolia* on incidence
746 of *E. harknessii* infection had been found in a previous multi-site study in British Columbia,
747 though thinning was associated with a large increase in the incidence of infection by
748 stalactiform blister rust (*Cronartium coleosporioides*) (van der Kamp, 1994). Similarly, in
749 Idaho, thinning was related to an increase in the number of new lethal infections per tree of
750 *Pinus monticola* (western white pine) by *C. ribicola* five years after treatment (Hungerford et
751 al., 1982). In an experiment in a forest in Missouri, USA, where a range of oak species are
752 subject to "oak decline" that may be caused by a range of root pathogens or insects, thinning
753 in the form of "improvement harvests" (selective cutting to remove trees that were declining
754 and to reduce tree density) did not significantly alter the incidence of oak decline after 10
755 (Meadows et al., 2013) or 14 years (Dwyer et al., 2007; Meadows et al., 2013).

756 As well as being a legacy of the harvesting of mature trees, stumps are also present
757 throughout growing stands as a result of thinning operations. Chemical or biological
758 treatment of stumps resulting from thinning can be effective at reducing pathogen incidence,
759 as is the case for final harvest tree stumps (section 4.3). In Sweden, following thinning, the
760 proportion of *P. abies* stump area colonised by *H. annosum* after 6-7 weeks was reduced by
761 88-99% in stumps treated with either 35% urea solution, 5% disodium octaborate tetrahydrate
762 solution or spores of *Phlebiopsis gigantea*, compared with untreated stumps (Thor and
763 Stenlid, 2005).

764 In the majority of cases, forests that have undergone thinning have a higher incidence of tree
765 disease than unthinned sites. However, results are variable, even within the same site,

766 pathogen or tree species. Such variation likely arises not from thinning itself, but from other
767 changes within the forest associated with thinning regimes. Pathogen loads can increase due
768 to increased movement of machinery and human vectors into a forest to carry out thinning
769 (Jules et al., 2002; Cushman and Meentemeyer, 2008; Goheen et al., 2012) and wounding of
770 stems and roots of retained trees that can provide entry points for pathogens. More complex
771 effects can be mediated by changes in forest species composition and structural composition
772 resulting from thinning. Therefore, to provide a more robust basis for management
773 recommendations, future studies should focus on identifying and accounting for the sources
774 of this variation, and determining how impacts vary during the course of a pathogen invasion.

775

776 **5.6 Diseased tree removal**

777 Removal of diseased trees is often one of the criteria applied for tree selection in thinning of
778 diseased stands. In some cases it is the sole focus of a control programme, either restricted to
779 trees already showing disease symptoms or extended to trees considered to be at high risk of
780 infection, e.g. because of their species and proximity to diseased trees. The effectiveness of
781 this measure has been assessed in a number of studies, though not through rigorous
782 experimentation. Examples include the spatial spread of Dutch elm disease (*Ophiostoma*
783 *novo*-ulmi) in New Zealand (Ganley and Bulman, 2016) and Gotland Island, Sweden
784 (Menkis et al., 2016), and *P. ramorum* in the coastal forests of Oregon (Kanaskie et al.,
785 2006). In 35-year-old coppiced *Castanea sativa* (sweet chestnut) in Italy, thinning that
786 targeted the cutting of infected stems did result in a reduction in the severity of damage due
787 to *C. parasitica* two years later (Amorini et al., 2001). In all of these studies, while some
788 evidence was found that removal of infected and adjacent trees slowed the spread of the
789 pathogen, it had only delayed, rather than prevented, eventual infection. The cryptic nature of
790 the pathogens that prevents sufficiently early identification of infected trees to enable their
791 removal before they become a source of inoculum, and the occasional occurrence of long-
792 distance inoculum dispersal events through vectors, e.g. human or animal movement, are
793 among the major constraints. The potential for removal of diseased trees to disrupt natural
794 biocontrol, e.g. hypovirulence of *C. parasitica* caused by virus infection of the fungus,
795 merits future research.

796

797 **5.7 Pruning and coppicing**

798 Pruning of lateral branches is usually carried out to improve timber quality by reducing knots
799 in the subsequent radial wood growth. Analogous to thinning, pruning may also be carried
800 out to reduce pathogen incidence by targeting infected or susceptible damaged branches or to
801 reduce sub-canopy humidity in the forest. However, pruning wounds also create potential
802 sites for pathogen entry and, as with thinning, pruning operations may increase traffic and
803 potential of cross-infection on tools, acting as vectors of pathogens.

804 Positive impacts of pruning *Pinus* spp. on resistance to *C. ribicola* rust have been largely
805 consistent across North America. In Idaho pruning of *P. monticola* in addition to thinning
806 greatly reduced the total number of new lethal and non-lethal infections per tree after five
807 years compared with thinning-only treatment and controls (Hungerford et al., 1982).
808 Incidence and severity of *C. ribicola* infection of young *P. strobus* was reduced by
809 preventative pruning of susceptible lower branches in sites across the eastern USA (Ostry,

2000). Pruning of infected branches of *P. strobus* also reduced the incidence of disease and tree mortality in Quebec, Canada (Lavallee, 1991).

For other pathogens, results of pruning have not been so positive. Pruning increased *F. circinatum* canker symptoms in *P. radiata* plantations in Cantabria, Spain, which was attributed to the role of pruning wounds in permitting the pathogen to infect the tree (Bezos et al., 2012). Pruning increased *D. pinea* infection in *P. radiata* trees in New Zealand that were experimentally inoculated, with a large increase in infection rates with intensity of pruning (percentage of crown removed) (Chou, 1988). Infection by *D. pinea* and by *Seiridium cardinale* was also positively associated with pruning of *Cupressus sempervirens* (cypress) trees in Israel (Madar et al., 1991). No effect of pruning was observed on control of *D. septosporum, D. pini* or *L. acicula* in *P. radiata* plantations in northern Spain (de Urbina et al., 2017), nor *Armillaria* spp. infection in New Zealand *P. radiata* plantations (Hood et al., 2002). For stands of *P. abies* in Baden-Württemburg, Germany, careful pruning of branches up to 10 m height was found to produce only a low risk of wood deterioration, however it did lead to an increase in heartwood infection by a range of pathogens, especially *Nectria fuckeliana* (Metzler, 1997). The review of studies in Australia and New Zealand on *Dothistroma* spp. infection of *Pinus* spp. by Bulman et al. (2016) reported mixed results of pruning, particularly beyond short-term impacts.

Shoot removal to reduce multiple stems to a single stem was carried out on *Acacia mangium* and *A. crassicarpa* plantation trees in South Africa, and was followed by experimental inoculation by pathogenic *Ceratocystis acaiivora* and *Lasiodiplodia theobromae* fungi (Tarigan et al., 2011). Careful pruning resulted in reduced lesion size compared with trees pruned less carefully, causing tearing of the bark, which suffered infection from naturally spreading spores even if not inoculated. Pathogen impacts are of particular concern for short-rotation coppice systems with fast-growing trees that are particularly susceptible to infection. In an experiment in Northern Ireland, McCracken and Dawson (2003) found that coppicing produced mixed results amongst genotypes of *Salix* spp. In one genotype, levels of the *M. epitea* rust pathogen were much higher in the first three-year harvest cycle than during the second cycle. However, for a number of other genotypes, *M. epitea* infection was more severe on the regrowth from freshly coppiced stools.

Phytophthora ramorum is able to persist in, and produce spores from, resprouted stumps (effectively increasing the inoculum load available for secondary infection within a plantation thus generating secondary infection). A benefit of sprout cutting was shown, as isolation of *P. ramorum* from the sprouts growing from cut stumps of *U. californica* was reduced in sprouts that had been cut one-year previously compared with those left to grow for seven years, however there was no treatment effect for sprouts growing on stumps of *N. densiflorus* (Valachovic et al., 2013a).

Pruning, coppicing and shoot removal have a highly variable impact on resilience of forests to tree pathogens. Some of the literature on the subject points to an increase in susceptibility caused by pruning wounds and increased vector or air movement of the pathogen within the forest. However, other studies show a decrease in susceptibility to some pathogens, linked to removal of susceptible branch material and reduced sub-canopy humidity. There is a lack of experimental studies that enable testing of these mechanisms and their trade-offs. While pruning is less common as a forest management practice than is thinning, it should be a priority for future studies. There is good potential to link knowledge of the effects of pruning practice on tree pathogens in arboriculture with the evidence required to inform forest

management. A priority is to understand more about what controls the risk of entry into pruning wounds of the main airborne pathogens of commercial tree species.

6 Recovery

The processes described above in terms of primary and secondary infection capture the first element of forest resilience, its resistance to an invading pathogen. The second element, the capacity of the forest to recover, is discussed in this section. As explained above, we considered rates of tree growth and natural regeneration following the onset of pathogen infection, which were the only measures of the recovery of the forest ecosystem reported in the reviewed studies. Within our working definition of resilience, we did not include changes in pathogen inoculum or infection level in the ecosystem as measures of recovery, in order to avoid mixing up "cause" and "effect". The capacity for forest ecosystem recovery can be assessed over a wide range of temporal and spatial scales. For entire managed forests it is extremely likely that, over the long term, the decisions of forest managers will be crucial in determining the rate and trajectory of forest recovery. Gibbs et al. (2002) provide an insightful account of how successive generations of managers have experimented and adapted the management of a forest (Thetford, UK) to promote recovery and longer-term forest resilience to the threat posed by *H. annosum*. A number of studies have investigated at a smaller and shorter-term scale the effect of individual management actions on the capacity for forest recovery from pathogen impacts through natural regeneration or tree growth. They have researched some of the forest management options already considered in terms of their impact on primary and secondary infection (above), and we review their evidence on forest recovery below, with a summary in Table 2.

6.1 Species diversity and mixtures

In a large experiment in British Columbia forests subject to infection by *A. ostoyae* and *P. sulphurascens*, mixed species plots had lower basal area after 40 years growth compared with monocultures, but the effect on stem diameter and on dominant tree height was variable (Morrison et al. 2014). Interpretation of other experimental studies in British Columbia crosses over between the effects of species mixture and of thinning as a treatment. In British Columbia forests where *P. menziesii* is subject to infection by *A. ostoyae*, mean diameter increment and height:diameter ratio increased significantly in stands where *B. papyrifera* was removed or partly thinned (Baleshta et al., 2005). This was linked to increases in light and soil moisture levels caused by the thinning. In higher altitude British Columbia forests infested with *A. ostoyae*, the diameter growth of planted *P. menziesii* was 27% greater after experimental removal of the naturally regenerated broadleaves *B. papyrifera* and *P. tremuloides* compared with untreated controls, and the increase was greater with higher intensity removal treatments, however height growth was not significantly affected (Gerlach et al., 1997; Baleshta et al., 2005; Simard et al., 2005). In a second experiment, diameter growth of *P. contorta* var. *latifolia* (but not *P. menziesii*) was increased by removal of broadleaves. These results indicate the importance of the identity of tree species in a mixture for tree growth recovery from pathogen infection.

In a complex mixture experiment of many *Salix* spp. varieties in a short rotation coppice system in Northern Ireland subject to the *M. epitea* rust pathogen, tree growth rate was

invariably greater in mixtures compared with monoculture, even when a majority of varieties in the mixture were killed by the pathogen (McCracken et al., 2001). However, in a subsequent experimental study of *Salix viminalis* genotypes subject to infection by *M. epitea*, whilst at the harvest at the end of the first three-year growth cycle mixtures showed a higher yield compared with monocultures, this difference did not persist to the harvest at the end of the second cycle (Begley et al., 2009).

6.2 Site preparation

Inoculum removal prior to planting has had mixed effects on subsequent tree growth. A large-scale experiment in a British Columbia forest infested with the root pathogens *P. sulphurascens* and *A. ostoyae* showed that stump removal increased plot basal area by an average of 1.3 times after 40 years of growth of the rotation following treatment, and increased dominant tree height, but did not alter stem diameter (Morrison et al., 2014). In forests infested by *P. sulphurascens* in Washington State and Oregon, pre-planting stump removal produced mixed results on the growth of *P. menziesii* (Thies and Westlind, 2005). It increased seedling height in two study sites, and reduced the final measured volume at one site, but there were no significant effects at the other sites studied.

6.3 Tree establishment under different silvicultural systems

There are insufficient studies of alternative silvicultural systems to draw any clear conclusions about the implications for recovery. In *P. radiata* forest infected by *F. circinatum* in California, gap size was positively associated with seedling height and diameter growth rates, showing a pattern that did not correspond to that of the variation in disease incidence with gap size (Ferchaw et al., 2013). In mixed conifer forest in Oregon subject to infection by *A. ostoyae*, experimental harvesting treatments of group selection and shelterwood were compared with unharvested forest (Filip *et al.* 2010). Diameter growth of retained trees ten years after infection by *A. ostoyae* was not significantly altered by the silvicultural harvesting treatments, and there were no consistent effects on the density of natural regeneration amongst species.

6.4 Tree density

In a *P. monticola* forest infected by *C. ribicola* in Washington State, tree height at 16 years of age was not significantly affected by tree spacing over a range from 3 to 5 m, however it was lower with very close spacing (2 m) and very wide spacing (6 m) (Bishaw et al., 2003). Tree diameter increased with spacing from 2 m to 5 m. Plot basal area and volume decreased with spacing over the whole range from 2 m to 6 m. Thus, response to variation in tree density differed amongst measures of forest growth. Results from this single study of one tree-pathogen species combination do not provide a sufficient basis for any generalisation.

6.5 Thinning

Studies of thinning impacts have consistently shown that it results in increased tree growth rates in infected stands sufficient to promote forest recovery. In *P. radiata* plantations in New Zealand subject to severe infection by *A. novae-zelandiae*, thinning treatments were followed by long-term increase in growth (and assumed associated resistance) in the retained trees at a level sufficient to counter the effects of increased inoculum potential following treatment (Hood & Kimberley 2009). As a result, it is expected that thinning of these diseased stands will not lead to any reduction in final crop volume. Similarly, in coppiced *C. sativa* forest infected by *C. parasitica* in Italy, a thinning treatment that targeted the cutting of infected stems increased the growth rate of the retained stems, resulting in the same stand volume growth rate as pre-thinning (Amorini et al., 2001). In Mississippi, USA, where red oaks (*Quercus* spp.) are infected with the canker decay fungus *Inonotus hispidus*, experimental improvement thinning that removed smaller and diseased trees significantly increased the diameter growth of the retained trees (Meadows et al. 2013).

One study was notable for providing evidence of natural regeneration as a process of forest recovery, but its results were mixed. In British Columbia selective cutting, a silvicultural treatment somewhat akin to thinning, in forests infested with *A. ostoyae*, resulted in a large increase in rates of subsequent natural regeneration of a range of conifer species, compared with uncut control plots, but in only two of the four studied forest sites. Less than 30% of the naturally regenerated trees were killed by the pathogen in these two sites (Morrison et al. 2001).

7 Conclusions

7.1 Coverage of published studies

Published studies on forest resilience to tree diseases have uneven coverage with regard to geographical locations, management options, and pathogen species. The majority of studies have been restricted to a single forest area, while larger-scale studies often find inconsistent results across locations. This patchy coverage, and a lack of detail in reporting of the management options tested or the scale of their effects, hampers our ability to produce a systematic assessment of the similarities and differences in the impacts of management on tree resilience to different tree pathogens. Insufficient evidence is provided to enable comparison of effectiveness between options. Individual studies are limited to considering a single, or small number of related, pathogens, and therefore do not provide an adequate evidence base for forest managers who need to decide how best to increase forest resilience against multiple known and unknown future threats. Determining general conclusions to best inform forest management in the face of such a diversity of (and likely increasing pressure from) future pathogen risks is therefore challenging. Most management actions have been responsive, seeking to combat specific pathogens that are either established in a forest or new outbreaks after they have reached a region. Interventions thus tend to focus on reducing sources of inoculum or the rate of secondary infection, including the transmission of inoculum and the susceptibility of trees. Because most studies have researched forests managed for timber production, their evidence should not simply be extrapolated to forests managed for other benefits (for which different measures of resilience, linked to other ecosystem services, would be more relevant).

7.2 Evidence of forest management that increases resilience to forest pathogens

Although the published studies included in this review were very uneven in their coverage and did not produce consistent results, they provided stronger evidence of the benefit of certain management options for forest resilience to pathogens. The reduction of primary infection by limiting the connectivity of forest units and by the removal or treatment of stumps during site preparation, and the reduction of secondary infection by planting mixed species forests, are the management options with the strongest evidence for improving forest resilience to pathogens. Despite this, in each case the effects are strongly modified by the particular methods used and tree species involved, so this evidence can only be taken as a first indication to inform management decision-making. Forest managers must also consider the scale of the effect of each management option and trade-offs with other impacts on the forest system, such as the effects on environmental conditions and thus tree health and vigor.

Commercial timber production is the dominant management objective in the studied forests, with the need to reduce the risk to this posed by tree pathogens and pests increasingly recognized. However, even in commercial forests biodiversity conservation is also an increasingly important objective. Therefore, the potential negative impacts of connectivity on risk of spread of pathogens and pests needs to be weighed against the demonstrated benefits of higher connectivity for biodiversity (Lindenmayer et al., 2006). For many tree pathogens, connectivity through vehicular and human movement may be more significant than a continuity of habitat cover, depending on how specific pathogens spread. Therefore, controlling primary infection rate via human vectors may provide a reduction in functional connectivity for some pathogens, with little impact on other organisms. Stump removal as a method to reduce sources of pathogen innocuum also presents an interesting trade-off with habitat management for the benefit of biodiversity. Dead wood such as stumps are recognised as important habitat components for a wide range of forest biodiversity, including many forest specialist species (Hartley, 2002). Providing adequate evidence to inform the management response to this trade-off is also a research priority.

Perhaps the most consistent finding of the reviewed research, supported by multiple studies across a range of pathogen species, although still not ubiquitous, is that the rate of secondary infection of trees of a given species is reduced if these trees are growing in a mixed, species-diverse forest rather than a monoculture. In this sense, higher species diversity has an insurance value against future income risks due to disease, which will be positively valued by risk-averse forest managers (Finger and Buchmann, 2015). However, this outcome depends on the species within the mixture, with only non-host species increasing forest resilience to pathogens. In fact, the presence of secondary host species can make infection worse and may increase the rate of primary infection (Power and Mitchell, 2004). This finding points to a need to extend the importance of tree species selection in strategies to combat tree diseases. Beyond considering the susceptibility of candidate primary crop species to pathogen species known to be present in the region (or at risk of arriving during the forest rotation), forest managers also need to consider the potential role of each tree species in a mixed forest for how it may influence the risk of infection (by a broad range of pathogens) of the other crop species present.

In seeking to achieve the most economically efficient solution to exploiting the benefits of higher tree species diversity for increasing forest system resilience to tree pathogens, a key consideration is the spatial scale at which such mixing occurs. If the economic benefit of

including within the forest a portfolio of different tree crop species can be achieved by establishing large monoculture blocks of each species, then this may only cause a small increase in management costs compared with a whole-forest monoculture. However, knowledge of ecological mechanisms would suggest that the larger the monoculture blocks the smaller will be the ecological benefit through diluting the individual trees of susceptible species. It is also possible that resilience of susceptible trees is increased due to interaction with other tree species, which is unlikely to occur if mixing takes place only at the landscape scale (Bauhus et al. 2017). It is therefore striking that we found no studies reporting results on the effect of the spatial scale of tree species mixing on rates of pathogen infection. Similarly, for the studies that researched at a landscape scale, we did not find any that reported the effects of the heterogeneity (e.g. in tree species composition) between adjacent forest patches. These evidence gaps limit our ability to draw detailed conclusions regarding how mixed-species forests should be designed to maximize this benefit, and we therefore identify these as high priorities for future research.

With reference to recovery of forest ecosystems, there are a number of studies of the effects of silvicultural treatments on the growth of mostly conifer crop trees in pathogen-infested forests. The results generally indicate that tree growth increased following silvicultural treatments, irrespective of the fact that the studies were carried out in forests where the trees were subject to pathogen infection. However, there are very few studies reporting on the effect of silvicultural treatments on forest recovery through natural regeneration.Implications for epidemiological and bioeconomic modelling

The findings of this review have several implications for epidemiological modelling of emergence, spread and persistence of tree pathogens and for capturing the resilience of forests in response to such threats. However, we identified many important evidence gaps in the empirical literature that should be a priority for new primary research to fill. Our review aimed at providing a foundation for linking the processes and parameters used in models, specifically the epidemiological components of primary and secondary infection, and the ecological components of forest recovery, to the published observational and experimental data. This has several important implications for this area of modelling. Firstly, while our review showed the importance of tree species mixture effects, most models consider a forest comprising only a single host species (Kleczkowski et al., 2019). Secondly, our review showed the importance of connectivity between forest units. While there has been significant progress in recent years on spatial and meta-population modelling of plant pathogens, to our knowledge these have not yet been combined with ecological models to improve understanding of the trade-offs between the benefits of connectivity for biodiversity (Lindenmayer et al., 2006) and the reduction of pathways for pathogens to spread between and within forests. Thirdly, in models the effects of management options on epidemiological processes are often described as a simple reduction of primary or secondary infection rate. In reality, many of these processes may involve nonlinearities and threshold behaviour, though these have not been thoroughly studied. Fourthly, our findings on the importance of persistent inoculum for certain pathogens, from reservoirs such as tree stumps and root fragments of felled trees, suggest that infection rates in a forest unit may be “path-dependent”, for example on the forest unit’s history of infection and control options adopted, with implications for the structure of models. Fifthly, the implications of the reviewed empirical studies for epidemiological modelling are limited by their generally small sample size and, in many cases, weakness in the capacity of the experimental design to test the influence of environmental variables and interaction effects.

We hope that this paper will contribute to a dialogue between forest managers and ecologists on one hand and epidemiological and bioeconomic modellers on the other, to establish criteria for experimentation that can be used to better parameterize models and rigorously test their results.

8 References

Amorini, E., Manetti, M.C., Turchetti, T., Sansotta, A., and Villani, F. (2001). Impact of silvicultural system on Cryphonectria parasitica incidence and on genetic variability in a chestnut coppice in Central Italy. *Forest Ecology and Management* 142(1-3)**,** 19-31. doi: 10.1016/s0378-1127(00)00337-6.

Anglberger, H., and Halmschlager, E. (2003). The severity of Sirococcus shoot blight in mature Norway spruce stands with regard to tree nutrition, topogrpahy and stand age. *Forest Ecology and Management* 177**,** 221-230.

Baleshta, K.E., Simard, S.W., Guy, R.D., and Chanway, C.R. (2005). Reducing paper birch density increases Douglas-fir growth rate and Armillaria root disease incidence in southern interior British Columbia. *Forest Ecology and Management* 208(1-3)**,** 1-13. doi: 10.1016/j.foreco.2004.07.076.

Bauhus, J., Forrester, D., Gardiner, B., Jactel, H., Vallejo, R., and Pretzsch, H. (2017). "Ecological stability of mixed-species forests," in *Mixed-Species Forests - Ecology and Management,* eds. H. Pretzsch, D.I. Forrester & J. Bauhus. (Verlag, Germany: Heidelberg), 337-382.

Bauman, J.M., Keiffer, C.H., and McCarthy, B.C. (2014). Growth performance and chestnut blight incidence (Cryphonectria parasitica) of backcrossed chestnut seedlings in surface mine restoration. *New Forests* 45(6)**,** 813-828. doi: 10.1007/s11056-014-9439-3.

Begley, D., McCracken, A.R., Dawson, W.M., and Watson, S. (2009). Interaction in Short Rotation Coppice willow, Salix viminalis genotype mixtures. *Biomass and Bioenergy* 33**,** 163-173.

Beh, M.M., Metz, M.R., Frangioso, K.M., and Rizzo, D.M. (2012). The key host for an invasive forest pathogen also facilitates the pathogen's survival of wildfire in California forests. *New Phytologist* 196(4)**,** 1145-1154. doi: 10.1111/j.1469-8137.2012.04352.x.

Bendz-Hellgren, M., and Stenlid, J. (1998). Effects of clear-cutting, thinning, and wood moisture content on the susceptibility of Norway spruce stumps to Heterobasidion annosum. *Canadian journal of forest research* 28(5)**,** 759-765.

Benedict (1981). History of white pine blister rust control - A personal account. *United States Department of Agriculture Forest Service*.

Bergdahl, D.R., Sendak, P.E., Tobi, D.R., Bove, J.R., and Tritton, L.M. (2002). Incidence of annual canker of sugar maple studied with the aid of a geographic information system. *Northern Journal of Applied Forestry* 19(2)**,** 73-79.

Bezos, D., Lomba, J.M., Martinez-Alvarez, P., Fernandez, M., and Diez, J.J. (2012). Effects of pruning in Monterrey pine plantations affected by Fusarium circinatum. *Forest Systems* 21(3)**,** 481-488. doi: 10.5424/fs/2012213-02262.

Bishaw, B., DeBell, D.S., and Harrington, C.A. (2003). Patterns of survival, damage, and growth for western white pine in a 16-year-old spacing trial in western Washington. *Western Journal of Applied Forestry* 18(1)**,** 35-43.

1118 Blodgett, J.T., Kruger, E.L., and Stanosz, G.R. (1997). Sphaeropsis sapinea and water stress in a red pine
1119 plantation in central Wisconsin. *Phytopathology* 87(4)**,** 429-434. doi:
1120 10.1094/phyto.1997.87.4.429.

1121 Boyd, I.L., Freer-Smith, P.H., Gilligan, C.A., and Godfray, H.C.J. (2014). The consequence of tree pests
1122 and diseases for ecosystem services. *Science* 342.

1123 Brassett, P.R., and Gilligan, C.A. (1988). A model for primary and secondary infection in botanical
1124 epidemics. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* 95**,** 352-360.

1125 Bulman, L.S. (2008). Pest detection surveys on high-risk sites in New Zealand. *Australian Forestry* 71(3)**,**
1126 242-244.

1127 Bulman, L.S., Bradshaw, R.E., Fraser, S., Martin-Garcia, J., Barnes, I., Musolin, D.L., et al. (2016). A
1128 worldwide perspective on the management and control of Dothistroma needle blight. *Forest*
1129 *Pathology* 46(5)**,** 472-488. doi: 10.1111/efp.12305.

1130 Burdon, J.J., Wennstrom, A., Ericson, L., Muller, W.J., and Morton, R. (1992). Density dependent
1131 mortality in Punis sylvestris cause by snow blight pathogen Phacidium infestans. *Oecologia*
1132 90(1)**,** 74-79.

1133 Campbell, E.A., JA. (2000). Distribution and severity of white pine blister rust and mountain pine beetle
1134 on whitebark pine in British Colombia. *Canadian Journal of Forest Research* 30**,** 1051-1059.

1135 Carey, M., Sanderson, B., Barnas, K., and Olden, J. (2012). Native invaders - challenges for science,
1136 management, policy, and society. *Frontiers in Ecology and the Environment* 10**,** 373-381.

1137 Chou, C.M., M. (1988). Effect of pruning intensity and season on Diplodia pinea infection of Pinus
1138 radiata stem through pruning wounds. *European Journal of Forest Pathology* 18**,** 437-444.

1139 Cleary, M.R., Arhipova, N., Morrison, D.J., Thomsen, I.M., Sturrock, R.N., Vasaitis, R., et al. (2013).
1140 Stump removal to control root disease in Canada and Scandinavia: A synthesis of results from
1141 long-term trials. *Forest Ecology and Management* 290**,** 5-14. doi:
1142 10.1016/j.foreco.2012.05.040.

1143 Condeso, T.M., RK (2007). Effects of landscape heterogeneity on the emerging forest disease sudden
1144 oak death. *Journal of Ecology* 95**,** 364-375.

1145 Cram, M.S., D. Forbus, K. (2010). Case study to examine the effects of a growing-season burn and
1146 Annosum root disease on mortality in a longleaf pine stand. *Proceedings of the 14th biennial*
1147 *souther silivicultural research conference*.

1148 Crone, M., McComb, J.A., O'Brien, P.A., and Hardy, G.E.S. (2014). Host removal as a potential control
1149 method for Phytophthora cinnamomi on severely impacted black gravel sites in the jarrah
1150 forest. *Forest Pathology* 44(2)**,** 154-159. doi: 10.1111/efp.12080.

1151 Cushman, J.H., and Meentemeyer, R.K. (2008). Multi-scale patterns of human activity and the
1152 incidence of an exotic forest pathogen. *Journal of Ecology* 96(4)**,** 766-776. doi: 10.1111/j.1365-
1153 2745.2008.01376.x.

1154 D'Souza, N.K., Colquhoun, I.J., Shearer, B.L., and Hardy, G.E.S. (2004). The potential of five Western
1155 Australian native Acacia species for biological control of Phytophthora cinnamomi. *Australian*
1156 *Journal of Botany* 52(2)**,** 267-279. doi: 10.1071/bt03089.

1157 Davidson, J.M., Patterson, H.A., Wickland, A.C., Fichtner, E.J., and Rizzo, D.M. (2011). Forest Type
1158 Influences Transmission of Phytophthora ramorum in California Oak Woodlands.
1159 *Phytopathology* 101(4)**,** 492-501. doi: 10.1094/phyto-03-10-0064.

de Urbina, E.O., Mesanza, N., Aragones, A., Raposo, R., Elvira-Recuenco, M., Boque, R., et al. (2017). Emerging Needle Blight Diseases in Atlantic Pinus Ecosystems of Spain. *Forests* 8(1). doi: 10.3390/f8010018.

Desprez-Loustau, M.L., Aguayo, J., Dutech, C., Hayden, K.J., Husson, C., Jakushkin, B., et al. (2016). An evolutionary ecology perspective to address forest pathology challenges of today and tomorrow. *Annals of Forest Science* 73(1)**,** 45-67. doi: 10.1007/s13595-015-0487-4.

Desprez-Loustau, M.L., and Wagner, K. (1997). Influence of silvicultural practices on twisting rust infection and damage in maritime pine, as related to growth. *Forest Ecology and Management* 98(2)**,** 135-147. doi: 10.1016/s0378-1127(97)00079-0.

Dillon, W.W., Haas, S.E., Rizzo, D.M., and Meentemeyer, R.K. (2014). Perspectives of spatial scale in a wildland forest epidemic. *European Journal of Plant Pathology* 138(3)**,** 449-465. doi: 10.1007/s10658-013-0376-3.

Dwyer, J.P., Kabrick, J.M., and Wetteroff, J. (2007). Do improvement harvests mitigate oak decline in Missouri Ozark forests? *Northern Journal of Applied Forestry* 24(2)**,** 123-128.

Ellis, A.M., Vaclavik, T., and Meentemeyer, R.K. (2010). When is connectivity important? A case study of the spatial pattern of sudden oak death. *Oikos* 119(3)**,** 485-493. doi: 10.1111/j.1600-0706.2009.17918.x.

Entry, J.A., Cromack, K. Kelsey, R.G. Martin, N.E. (1991). Response of Douglas-Fir to Infection by Armillaria ostoyae After Thinning or Thinning Plus Fertilisation. *Phytopathogy*.

Ferchaw, V.A.L., Goldsworthy, E., Pinkerton, J., Yun, D.I., Lund, U.J., Mark, W., et al. (2013). Management strategies for pitch canker infected Ano Nuevo stands of Monterey pine. *Forest Ecology and Management* 308**,** 101-115. doi: 10.1016/j.foreco.2013.07.032.

Fichtner, E.J., Rizzo, D.M., Kirk, S.A., and Webber, J.F. (2011). Root Infections May Challenge Management of Invasive Phytophthora spp. in UK Woodlands. *Plant Disease* 95(1)**,** 13-18. doi: 10.1094/pdis-03-10-0236.

Filip, G.M., Ganio, L.M., Oester, P.T., Mason, R.R., and Wickman, B.E. (2002). Ten-year effect of fertilization on tree growth and mortality associated with armillaria root disease, fir engravers, dwarf mistletoe, and western spruce budworm in Northeastern Oregon. *Western Journal of Applied Forestry* 17(3)**,** 122-128.

Filip, G.M., Maffei, H.M., Chadwick, K.L., and Max, T.A. (2010). Armillaria Root Disease-Caused Tree Mortality following Silvicultural Treatments (Shelterwood or Group Selection) in an Oregon Mixed-Conifer Forest: Insights from a 10-Year Case Study. *Western Journal of Applied Forestry* 25(3)**,** 136-143.

Finger, R., and Buchmann, N. (2015). An ecological economic assessment of risk-reducing effects of species diversity in managed grasslands. *Ecological Economics* 110(89-97).

Ganley, R.J., and Bulman, L.S. (2016). Dutch elm disease in New Zealand: impacts from eradication and management programmes. *Plant Pathology* 65(7)**,** 1047-1055. doi: 10.1111/ppa.12527.

Gerlach, J.P., Reich, P.B., Puettmann, K., and Baker, T. (1997). Species, diversity, and density affect tree seedling mortality from Armillaria root rot. *Canadian Journal of Forest Research-Revue Canadienne De Recherche Forestiere* 27(9)**,** 1509-1512. doi: 10.1139/cjfr-27-9-1509.

Ghelardini, L., Luchi, N., Pecori, F., Pepori, A.L., Danti, R., Della Rocca, G., et al. (2017). Ecology of invasive forest pathogens. *Biological Invasions* 19(11)**,** 3183-3200. doi: 10.1007/s10530-017-1487-0.

Gibbs, J.N., Greig, B.J.W., Pratt, J.E. (2002). Fromes root rot in Thetford Forest, East Anglia: past, present and future. *Forestry* 75(2)**,** 191-202.

1205 Goheen, D.J., Mallams, K., Betlejewski, F., and Hansen, E. (2012). Effectiveness of Vehicle Washing and
1206 Roadside Sanitation in Decreasing Spread Potential of Port-Orford-Cedar Root Disease.
1207 *Western Journal of Applied Forestry* 27(4)**,** 170-175. doi: 10.5849/wjaf.11-011.

1208 Greene, T.A., Reemts, C.M., and Appel, D.N. (2008). Efficacy of Basal Girdling to Control Oak Wilt
1209 Fungal Mat Production in Texas Red Oak (Quercus buckleyi) in Central Texas. *Southern Journal*
1210 *of Applied Forestry* 32(4)**,** 168-172.

1211 Grimm, V., and Wissel, C. (1997). Babel, or the ecological stability discussions: an inventory and
1212 analysis of terminology and a guide for avoiding confusion. *Oecologia* 109**,** 323-334.

1213 Haas, S.E., Cushman, J.H., Dillon, W.W., Rank, N.E., Rizzo, D.M., and Meentemeyer, R.K. (2016). Effects
1214 of individual, community, and landscape drivers on the dynamics of a wildland forest
1215 epidemic. *Ecology* 97(3)**,** 649-660. doi: 10.1890/15-0767.1.

1216 Haas, S.E., Hooten, M.B., Rizzo, D.M., Meetenmeyer, R.K. (2011). Forest species diversity reduces
1217 disease risk in a generalist plant pathogen invasion. *Ecology Letters* 14**,** 1108 - 1116.

1218 Hagle, S.K., Grasham, J.L. (1988). Biological and economic feasibility of pruning and excising white
1219 pines for blister rust control. *United States Department of Agriculture Forest Service*.

1220 Halmschlager, E., and Katzensteiner, K. (2017). Vitality fertilisation balanced tree nutrition and
1221 mitigated severity of Sirococcus shoot blight on mature Norway spruce. *Forest Ecology and*
1222 *Management* 389**,** 96-104.

1223 Hannerz, M., and Hanell, B. (1997). Effects on the flora in Norway spruce forests following clearcutting
1224 and shelterwood cutting. *Forest Ecology and Management* 90**,** 29-49.

1225 Hantsch, L., Bien, S., Radatz, S., Braun, U., Auge, H., and Bruelheide, H. (2014a). Tree diversity and the
1226 role of non-host neighbour tree species in reducing fungal pathogen infestation. *Journal of*
1227 *Ecology* 102(6)**,** 1673-1687. doi: 10.1111/1365-2745.12317.

1228 Hantsch, L., Braun, U., Haase, J., Purschke, O., Scherer-Lorenzen, M., and Bruelheide, H. (2014b). No
1229 plant functional diversity effects on foliar fungal pathogens in experimental tree communities.
1230 *Fungal Diversity* 66(1)**,** 139-151. doi: 10.1007/s13225-013-0273-2.

1231 Hantsch, L.B., U. Scherer-Lorenzen, M. Bruelheide, H. (2013). Species richness and species identity
1232 effects on occurrence of foliar fungal pathogens in a tree diversity experiment. *Ecosphere* 4(7).

1233 Harrington, T.C., Reinhart, C., Thornburgh, D.A., and Cobb Jr, F.W. (1983). Association of black-stain
1234 root disease with precommercial thinning of Douglas-fir. *Forest Science* 29(1)**,** 12-14.

1235 Hartley, M.J. (2002). Rationale and methods for conserving biodiversity in plantation forests. *Forest*
1236 *Ecology and Management* 155**,** 81-95.

1237 Havdova, L.Z., D. Romportl, D. Peskova, V. Cerny, K. (2017). Environmental and silvicultural
1238 characteristics influencing the extent of ash diebakc in forest stands. *Baltic Forestry* 23(1)**,**
1239 168-182.

1240 Hessburg, P. (2001). Association of black stain root disease with roads, skid trails, and precommercial
1241 thinning in southwest Oregon. *Western Journal of Applied Forestry* 16(3)**,** 127-135.

1242 Heybroek, H.M. (1982). Monoculture versus mixture: interactions between susceptible and resistant
1243 trees in a mixed stand. *Proceedings the third international workshop on the genetics of Host-*
1244 *Parasite interactions in forestry, Wageningen, the Netherlands*.

1245 Hill, L., Jones, G., Atkinson, N., Hector, A., Hemery, G., and Brown, N. (2019). The £15 billion cost of
1246 ask dieback in Britian. *Current Biology* 29(9)**,** 315-316.

1247 Holling, C.S. (1996). "Engineering resilience versus ecological resilience," in *Engineering within*
1248 *Ecological Constraints,* ed. P.C. Schulze. (Washington DC: National Academy Press), 31-43.

1249 Holzmueller, E.J., Jose, S., and Jenkins, M.A. (2008). The relationship between fire history and an exotic
1250 fungal disease in a deciduous forest. *Oecologia* 155(2)**,** 347-356. doi: 10.1007/s00442-007-
1251 0908-7.

1252 Hood, I.A., and Kimberley, M.O. (2009). Impact of armillaria root disease and the effect of thinning in
1253 a late-rotation Pinus radiata plantation. *Forest Pathology* 39(6)**,** 415-427. doi: 10.1111/j.1439-
1254 0329.2009.00602.x.

1255 Hood, I.A., Kimberley, M.O., Gardner, J.F., and Sandberg, C.J. (2002). Armillaria root disease of Pinus
1256 radiata in New Zealand. Influence of thinning and pruning. *New Zealand Journal of Forestry*
1257 *Science* 32(1)**,** 116-132.

1258 Hungerford, R.D., Williams, R.E., and Marsden, M.A. (1982). Thinning and pruning Western white pine:
1259 A potential for reducing mortality due to blister rust. *United States Department of Agriculture*
1260 *Forest Service*.

1261 Hunt, R.S., Geils, B.W., and Hummer, K.E. (2010). White pines, Ribes, and blister rust: integration and
1262 action. *Forest Pathology* 40(3-4)**,** 402-417. doi: 10.1111/j.1439-0329.2010.00665.x.

1263 Jactel, H., Bauhus, J., Boberg, J., Bonal, D., Castagneyrol, B., Gardiner, B., et al. (2017). Tree Diversity
1264 Drives Forest Stand Resistance to Natural Disturbances. *Current Forestry Reports* 3(3)**,** 223-
1265 243. doi: 10.1007/s40725-017-0064-1.

1266 Jactel, H., Nicoll, B.C., Branco, M., Gonzalez-Olabarria, J.R., Grodzki, W., Langstrom, B., et al. (2009).
1267 The influences of forest stand management on biotic and abiotic risks of damage. *Annals of*
1268 *Forest Science* 66(7). doi: 10.1051/forest/2009054.

1269 Jules, E.S., Kauffman, M.J., Ritts, W.D., and Carroll, A.L. (2002). Spread of an invasive pathogen over a
1270 variable landscape: A nonnative root rot on Port Orford Cedar. *Ecology* 83(11)**,** 3167-3181.

1271 Kaitera, J. (2002). Short-term Effect of Thinning on Pinus sylvestris Damage and Sporulation Caused by
1272 Cronartium flaccidum. *Scandinavian Journal of Forest Research* 17(2)**,** 158-165. doi:
1273 10.1080/028275802753626809.

1274 Kanaskie, A., Osterbauer, N., McWilliams, M., Goheen, E., Hansen, E., and Sutton, W. (2006).
1275 Eradication of Phytophthora ramorum in Oregon forests - Status after 3 years. *Proceedings of*
1276 *the sudden oak death second science symposium: the state of our knowledge*.

1277 Katovich, S., O'Brien, J.G., Mielke, M.E., and Ostry, M. (2004). Restoration and management of Eastern
1278 white pine within high blister rust hazard zones in the lake states. *USDA Forest Service*
1279 *Proceeding***,** 135-145.

1280 King, D.I., and DeGraaf, R.M. (2000). Bird species diversity and nesting success in mature, clearcut and
1281 shelterwood forest in northern New Hampshire, USA. *Forest Ecology and Management* 129**,**
1282 227-235.

1283 Kleczkowski, A., Hoyle, A., and McMenemy, P. (2019). One model to rule them all? Modelling
1284 approaches across OneHealth for human, animal and plant epidemics. *Philosophical*
1285 *Transactions of the Royal Society B* 374.

1286 Kwon, T.S., Shin, J.H., Lim, J.H., Kim, Y.K., and Lee, E.J. (2011). Management of pine wilt disease in
1287 Korea through preventative silvicultural control. *Forest Ecology and Management* 261(3)**,** 562-
1288 569. doi: 10.1016/j.foreco.2010.11.008.

1289 Lavallee, A. (1991). White pine blister rust control in a 5 year old eastern white pine plantation at
1290 Vercheres, Quebec (1984-88 results). *Proceedings of the IUFRO rusts of pine working party*
1291 *conference*.

1292 Leak, W.B. (2006). Fifty-year impacts of the beech bark disease in the Bartlett Experimental Forest,
1293 New Hampshire. *Northern Journal of Applied Forestry* 23(2)**,** 141-143.

Liebhold, A.M., Brockerhoff, E.G., Kalisz, S., Nuñez, M.A., Wardle, D.A., and Wingfield, M.J. (2017). Biological invasions in forest ecosystems. *Biological Invasions*. doi: 10.1007/s10530-017-1458-5.

Linden, M.L., and Vollbrecht, G. (2002). Sensitivirt of Picea abies to Butt Rot in pure stands and mixed stands with Pinus sylvestric in Southern Sweden. *Silva Fennica* 36(4)**,** 767-778.

Lindenmayer, D.B., Franklin, J.F., and Fischer, J. (2006). General management principles and a checklist of strategies to guide forest biodiversity conservation. *Biological Conservation* 131(3)**,** 433-445. doi: 10.1016/j.biocon.2006.02.019.

Macpherson, M.F., Kleczkowski, A., Healey, J.R., and Hanley, N. (2017a). Payment for multiple forest benefits alters the effect of tree disease on optimal forest rotation length. *Ecological Economics* 134**,** 82-94. doi: 10.1016/j.ecolecon.2017.01.008.

Macpherson, M.F., Kleczkowski, A., Healey, J.R., and Hanley, N. (2018). The Effects of Disease on Optimal Forest Rotation: A Generalisable Analytical Framework. *Environmental and Resource Economics* 70(3)**,** 565-588. doi: 10.1007/s10640-016-0077-4.

Macpherson, M.F., Kleczkowski, A., Healey, J.R., Quine, C.P., and Hanley, N. (2017b). The effects of invasive pests and pathogens on strategies for forest diversification. *Ecological Modelling* 350**,** 87-99. doi: 10.1016/j.ecolmodel.2017.02.003.

Madar, Z., Solel, Z., Sztejnberg, A., and Kimchi, M. (1991). EFFECT OF PRUNING OF TRUNK SIDE-BRANCHES OF CYPRESS ON INFECTION BY SEIRIDIUM-CARDINALE AND DIPLODIA-PINEA F-SP CUPRESSI. *Forest Ecology and Management* 44(2-4)**,** 255-260. doi: 10.1016/0378-1127(91)90012-k.

Martin, J., Cabezas, J., Buyolo, T., and Paton, D. (2005). The relationship between Cerambyx spp. damage and subsequent Biscogniauxia mediterranum infection on Quercus suber forests. *Forest Ecology and Management* 216(1-3)**,** 166-174. doi: 10.1016/j.foreco.2005.05.027.

Matsushita, K., Taki, H., Yamane, F., and Asano, K. (2018). Shadow Value of Ecosystem Resilience in Complex Natural Land as a Wild Pollinator Habitat. *American Journal of Agricultural Economics* 100**,** 829-843.

Mattila, U. (2002). The risk of pine twisting rust damage in young Scots pines: a multilevel logit model approach. *Forest Ecology and Management* 165(1-3)**,** 151-161. doi: 10.1016/s0378-1127(01)00657-0.

Mattila, U., Jalkanen, R., and Nikula, A. (2001). The effects of forest structure and site characteristics on probability of pine twisting rust damage in young Scots pine stands. *Forest Ecology and Management* 142(1-3)**,** 89-97. doi: 10.1016/s0378-1127(00)00342-x.

McCracken, A.R., and Dawson, W.M. (1997). Growing clonal mixtures of willow to reduce effect of Melampsora epitea var. epitea. *European Journal of Forest Pathology* 27**,** 319-329.

McCracken, A.R., and Dawson, W.M. (1998). Short rotation coppic willow in Northern Ireland since 1973: development of the use of mixtures in the control of foliar rust (Melampsora spp.). *European Journal of Forest Pathology* 28**,** 241-250.

McCracken, A.R., and Dawson, W.M. (2003). Rust disease (Melamspora epitea) of willow (Salix spp.) grown in short rotation coppice (SRC) in inter- and intra-species mixtures. *Annals of Applied Biology* 143**,** 381-393.

McCracken, A.R., Dawson, W.M., and Bowden, G. (2001). Yield response of willow (Salix) grown in mixtures in short rotation coppice (SRC). *Biomass & Bioenergy* 21**,** 311-319.

Meadows, J.S., Leininger, T.D., Montwe, D., and Nebeker, T.E. (2013). Thinning to improve growth, bole quality, and forest health in an Inonotus hispidus infected red oak-sweetgum stand in the

Mississippi delta: 10 year results. *Proceedings of the 15th biennial southern silvicultural research conference*.

Meentemeyer, R.K., Rank, N.E., Anacker, B.L., Rizzo, D.M., and Cushman, J.H. (2008a). Influence of land-cover change on the spread of an invasive forest pathogen. *Ecological Applications* 18(1)**,** 159-171. doi: 10.1890/07-0232.1.

Meentemeyer, R.K., Rank, N.E., Shoemaker, D.A., Oneal, C.B., Wickland, A.C., Frangioso, K.M., et al. (2008b). Impact of sudden oak death on tree mortality in the Big Sur ecoregion of California. *Biological Invasions* 10(8)**,** 1243-1255. doi: 10.1007/s10530-007-9199-5.

Menkis, A., Ostbrant, I.L., Wagstrom, K., and Vasaitis, R. (2016). Dutch elm disease on the island of Gotland: monitoring disease vector and combat measures. *Scandinavian Journal of Forest Research* 31(3)**,** 237-241. doi: 10.1080/02827581.2015.1076888.

Metzler, B. (1997). Quantitative assessment of fungal colonisation in Norway spruce after green pruning. *European Journal of Forest Pathology* 27**,** 1-11.

Millar, C.I., and Stephenson, N.L. (2015). Temperate forest health in an era of emerging megadisturbance. *Science* 349(6250)**,** 823-826.

Morrison, D.J., Cruickshank, M.G., and Lalumiere, A. (2014). Control of laminated and Armillaria root diseases by stump removal and tree species mixtures: Amount and cause of mortality and impact on yield after 40 years. *Forest Ecology and Management* 319**,** 75-98. doi: 10.1016/j.foreco.2014.02.007.

Morrison, D.J., Pellow, K.W., Nemec, A.F.L., Norris, D.J., and Semenoff, P. (2001). Effects of selective cutting on the epidemiology of armillaria root disease in the southern interior of British Columbia. *Canadian Journal of Forest Research* 31(1)**,** 59-70. doi: 10.1139/cjfr-31-1-59.

Morrison, D.J., Wallis, G.W., and Weir, L.C. (1988). Control of Armillaria and Phellinus root diseases: 20 year results from the Skimikin stump removal experiment. *Canadian Forestry Service*.

Muzika, R.M. (2017). Opportunities for silviculture in management and restoration of forests affected by invasive species. *Biological Invasions*. doi: 10.1007/s10530-017-1549-3.

Nebeker, T.E., Leininger, T.D., and Meadows, J.S. (1999). Silvicultural practices in forests of the Southern United States: Insect and disease considerations. *Tenth Biennial Southern Silvicultural Research Conference*.

Newton, A.C., and Cantarello, E. (2015). Restoration of forest resilience: an achievable goal? *New Forests* 46(5-6)**,** 645-668.

Nguyen, T.P.L., Wright, E.P., Nguyen, T.T., Schuiling-Veninga, C.C.M., Bijlsma, M.J., Nguyen, T.B.Y., et al. (2016). Cost-Effectiveness Analysis of Screening for and Managing Identified Hypertension for Cardiovascular Disease Prevention in Vietnam. *Plos One* 11(5). doi: 10.1371/journal.pone.0155699.

Nguyen, T.V., Park, Y.S., Jeoung, C.S., Choi, W.I., Kim, Y.K., Jung, I.H., et al. (2017). Spatially explicit model applied to pine wilt disease dispersal based on host plant infestation. *Ecological Modelling* 353**,** 54-62. doi: 10.1016/j.ecolmodel.2016.10.022.

Oliva, J., Samils, N., Johansson, U., Bendz-Hellgren, M., and Stenlid, J. (2008). Urea treatment reduced Heterobasidion annosum sl root rot in Picea abies after 15 years. *Forest ecology and management* 255(7)**,** 2876-2882.

Oliva, J., Suz, L.M., and Colinas, C. (2009). Ecology of Armillaria species on silver fir (Abies alba) in the Spanish Pyrenees. *Annals of Forest Science* 66(6). doi: 10.1051/forest/2009046.

Oliva, J., Thor, M., and Stenlid, J. (2010). Long-term effects of mechanized stump treatment against Heterobasidion annosum root rot in Picea abies. *Canadian Journal of Forest Research* 40(6)**,** 1020-1033.

Ostry, M. (2000). Restoration of White Pine in Minnesota, Wisconsin, and Michigan. *Hort Techology* 10(3)**,** 542-543.

Ostry, M.E., Laflamme, G., and Katovich, S.A. (2010). Silvicultural approaches for management of eastern white pine to minimize impacts of damaging agents. *Forest Pathology* 40(3-4)**,** 332-346. doi: 10.1111/j.1439-0329.2010.00661.x.

Ostry, M.E., Moore, M.J., Kern, C.C., Venette, R.C., and Palik, B.J. (2012). Multiple diseases impact survival of pine species planted in red pine stands harvested in spatially variable retention patterns. *Forest Ecology and Management* 286**,** 66-72. doi: 10.1016/j.foreco.2012.08.017.

Otrosina, W.J., Kliejunas, J.T., Smith, S., Cluck, D.R., Sung, S.S., and Cook, C.D. (2007). Black stain root disease studies on Ponderosa pine- Parameters and disturbanc treatments affecting infection and mortality. *Acta Silva. Lign. Hung., Spec. Edition***,** 247-251.

Pankuch, J.M., Blenis, P.V., Lieffers, V.J., and Mallett, K.I. (2003). Fungal colonization of aspen roots following mechanical site preparation. *Canadian Journal of Forest Research-Revue Canadienne De Recherche Forestiere* 33(12)**,** 2372-2379. doi: 10.1139/x03-172.

Pautasso, M., Aas, G., Queloz, V., and Holdenrieder, O. (2013). European ash (Fraxinus excelsior) dieback - A conservation biology challenge. *Biological Conservation* 158**,** 37-49. doi: 10.1016/j.biocon.2012.08.026.

Pautasso, M., Holdenrieder, O., and Stenlid, J. (2005). Susceptibility to fungal pathogens of forests differing in tree diversity. *Forest diversity and function*.

Peacock, L., Hunter, T., Turner, H., and Brain, P. (2001). Does host genotype diversity affect the distribution of insect and disease damage in willow cropping systems? *Journal of Applied Ecology* 38(5)**,** 1070-1081. doi: 10.1046/j.1365-2664.2001.00655.x.

Peterson, E., Hansen, E., and Kanaskie, A. (2014). Spatial relationship between Phytophthora ramorum and roads or streams in Oregon tanoak forests. *Forest Ecology and Management* 312**,** 216-224. doi: 10.1016/j.foreco.2013.10.002.

Piirto, D.D., and Valkonen, S. (2005). Structure and development of pitch canker infected Monterey pine stands at Ano Nuevo, California. *Forest Ecology and Management* 213(1-3)**,** 160-174. doi: 10.1016/j.foreco.2005.03.026.

Pimm, S.L. (1984). The complexity and stability of ecosystems. *Nature* 307**,** 321-326.

Pleysier, C.E., Bayliss, K.L., Dell, B., and Hardy, G.E.S. (2006). Temperature, humidity, wounding and leaf age influence the development of Alternaria alternata lesions on leaves of Paulownia fortunei. *Australasian Plant Pathology* 35(3)**,** 329-333. doi: 10.1071/ap06030.

Power, A.G., and Mitchell, C.E. (2004). Pathogen spillover in disease epidemics. *American Naturalist* 164**,** S79-S89.

Poyatos, R., Latron, J., and Llorens, P. (2002). Land Use and Land Cover Change After Agricultural Abandonment. *Mountain Research and Development* 23(4)**,** 362-368.

Prospero, S., and Cleary, M. (2017). Effects of Host Variability on the Spread of Invasive Forest Diseases. *Forests* 8(3). doi: 10.3390/f8030080.

Puddu, A., Luisi, N., Capretti, P., and Santini, A. (2003). Environmental factors related to damage by Heterobasidion abietinum in Abies alba forests in Southern Italy. *Forest Ecology and Management* 180(1-3)**,** 37-44. doi: 10.1016/s0378-1127(02)00607-2.

Ramage, B.S., Forrestel, A.B., Moritz, M.A., and O'Hara, K.L. (2012). Sudden oak death disease progression across two forest types and spatial scales. *Journal of Vegetation Science* 23(1), 151-163. doi: 10.1111/j.1654-1103.2011.01340.x.

Raymond, P., and Bédard, S. (2017). The irregular shelterwood system as an alternative to clearcutting to achieve compositional and structural objectives in temperate mixedwood stands. *Forest Ecology and Management* 398, 91-100. doi: 10.1016/j.foreco.2017.04.042.

Richter, B.S., Benson, D.M., and Ivors, K.L. (2011a). Microbial Profiling of Cultural Systems for Suppression of Phytophthora Root Rot in Fraser Fir. *Plant Disease* 95(5), 537-546. doi: 10.1094/pdis-03-10-0238.

Richter, B.S., Ivors, K., Shi, W., and Benson, D.M. (2011b). Cellulase Activity as a Mechanism for Suppression of Phytophthora Root Rot in Mulches. *Phytopathology* 101(2), 223-230. doi: 10.1094/phyto-04-10-0125.

Roach, W.J., Simard, S.W., and Sachs, D.L. (2015). Evidence against planting lodgepole pine monocultures in the cedar-hemlock forests of southeastern British Columbia. *Forestry* 88(3), 345-358. doi: 10.1093/forestry/cpv005.

Robinson, R.M. (2003). Short-term impact of thinning and fertilizer application on Armillaria root disease in regrowth karri (Eucalyptus diversicolor F. Muell.) in Western Australia. *Forest Ecology and Management* 176(1-3), 417-426. doi: 10.1016/s0378-1127(02)00231-1.

Ronnberg, J. (2000). Logging operation damage to roots of clear-felled Picea abies and Subsequent spore infection by Heterobasidion annosum. *Silva Fennica* 34(1), 29-36.

Rosenvald, R., Drenkhan, R., Riit, T., and Lohmus, A. (2015). Towards silvicultural mitigation of the European ash (Fraxinus excelsior) dieback: the importance of acclimated trees in retention forestry. *Canadian Journal of Forest Research* 45(9), 1206-1214. doi: 10.1139/cjfr-2014-0512.

Rosso, P., and Hansen, E. (1998). Tree vigour and the susceptibility of Douglas fir to Armillaria root disease. *Forest Pathology* 28(1), 43-52. doi: 10.1111/j.1439-0329.1998.tb01164.x.

Sakamoto, K., Tamesa, T., Yukio, T., Tokuhisa, Y., Maeda, Y., and Oka, M. (2016). Risk Factors and Managements of Bile Leakage After Hepatectomy. *World Journal of Surgery* 40(1), 182-189. doi: 10.1007/s00268-015-3156-8.

Santini, A., Pepori, A., Ghelardini, L., and Capretti, P. (2008). Persistence of some pine pathogens in coarse woody debris and cones in a Pinus pinea forest. *Forest Ecology and Management* 256(3), 502-506. doi: 10.1016/j.foreco.2008.05.010.

Schoettle, A.W., and Sniezko, R.A. (2007). Proactive intervention to sustain high-elevation pine ecosystems threatened by white pine blister rust. *Journal of Forest Research* 12(5), 327-336. doi: 10.1007/s10310-007-0024-x.

Shaw, C.G., Omdal, D.W., Ramsey-Kroll, A., and Roth, L.F. (2012). Inoculum Reduction Measures to Manage Armillaria Root Disease in a Severely Infected Stand of Ponderosa Pine in South-Central Washington: 35-Year Results. *Western Journal of Applied Forestry* 27(1), 25-29.

Simard, S.W., Hagerman, S.M., Sachs, D.L., Heineman, J.L., and Mather, W.J. (2005). Conifer growth, Armillaria ostoyae root disease, and plant diversity responses to broadleaf competition reduction in mixed forests of southern interior British Columbia. *Canadian Journal of Forest Research* 35(4), 843-859. doi: 10.1139/x05-010.

Staudhammer, C.L., Jokela, E.J., and Martin, T.A. (2009). Competition dynamics in pure- versus mixed-family stands of loblolly and slash pine in the southeastern United States. *Canadian Journal of Forest Research-Revue Canadienne De Recherche Forestiere* 39(2), 396-409. doi: 10.1139/x08-184.

1471 Swedjemark, G., and Stenlid, J. (1993). Population dynamics of the root rot fungus Heterobasidion
1472 annosum following thinning of Picea abies. *Oikos*, 247-254.

1473 Tarigan, M., Wingfield, M.J., van Wyk, M., Tjahjono, B., and Roux, J. (2011). Pruning quality affects
1474 infection of Acacia mangium and A. crassicarpa by Ceratocystis acaciivora and Lasiodiplodia
1475 theobromae. *Southern Forests* 73(3-4), 187-191. doi: 10.2989/20702620.2011.639498.

1476 Thies, W.G., Kelsey, R.G., Westlind, D.J., and Madsen, J. (2006). Potassium fertilizer applied
1477 immediately after planting had no impact on Douglas-fir seedling mortality caused by
1478 laminated root rot on a forested site in Washington State. *Forest Ecology and Management*
1479 229(1-3), 195-201. doi: 10.1016/j.foreco.2006.03.028.

1480 Thies, W.G., and Westlind, D.J. (2005). Stump removal and fertilization of five Phellinus weirii-infested
1481 stands in Washington and Oregon affect mortality and growth of planted Douglas-fir 25 years
1482 after treatment. *Forest Ecology and Management* 219(2-3), 242-258. doi:
1483 10.1016/j.foreco.2005.08.050.

1484 Thor, M., and Stenlid, J. (2005). Heterobasidion annosuminfection ofPicea abiesfollowing manual or
1485 mechanized stump treatment. *Scandinavian Journal of Forest Research* 20(2), 154-164. doi:
1486 10.1080/02827580510008338.

1487 Valachovic, Y., Cobb, R.C., Rizzo, D., Twieg, B., Lee, C., and Glebocki, R. (2013a). Is stump sprout
1488 treatment necessary to effectively control Phytophthora ramorum in California;s wildlands.
1489 *Proceedings of the sudden oak death fifth science symposium*.

1490 Valachovic, Y., Lee, C., Marshall, J., and Scanlon, H. (2010). "Forest Treatment Strategies for
1491 Phytophthora ramorum," in *Proceedings of the Sudden Oak Death Fourth Science*
1492 *Symposium*.), 239-+.

1493 Valachovic, Y., Lee, C., Twieg, B., Rizzo, D., Cobb, R.C., and Glebocki, R. (2013b). Suppression of
1494 Phytophthora ramorum infestation through silvicultural treatment in California's north coast.
1495 *Proceedings of the sudden oak death fifth science symposium*.

1496 van der Kamp, B.J. (1994). Lodgepole pine stem disease and management of stand density in the
1497 British Colombia interior. *The Forestry Chronicle* 70(6), 773-779.

1498 Walker, B., Pearson, L., Harris, M., Maler, K.G., Li, C., Biggs, R., et al. (2010). Incorporating resilience in
1499 the assessment of inclusive wealth. *Environmental and Resource Economics* 45, 183-202.

1500 Ward, J.S., Anagnostakis, S., and Ferrandino, F.J. (2010). Nectria Canker Incidence on Birch (Betula
1501 spp.) in Connecticut. *Northern Journal of Applied Forestry* 27(3), 85-91.

1502 Waring, K.M., and O'Hara, K.L. (2005). Silvicultural strategies in forest ecosystems affected by
1503 introduced pests. *Forest Ecology and Management* 209(1-2), 27-41. doi:
1504 10.1016/j.foreco.2005.01.008.

1505 Warren, C. (2007). Perspectives on the 'alien' vs 'native' species debate: a critique of concepts,
1506 language and practice. *Progress in Human Geography* 31(4), 427-446.

1507 Whitney, R.D., and Irwin, R.N. (2005). Comparison of Armillaria root disease on burned and unburned,
1508 harvested sites in Ontario. *Forestry Chronicle* 81(1), 56-60.

1509 Wingfield, M.J., Brockerhoff, E., Wingfield, B.D., and Slippers, B. (2015). Planted forest health: The
1510 need for a global strategy. *Science* 349(6250), 832-836.

1511 Wingfield, M.J., Slippers, B., Wingfield, B.D., and Barnes, I. (2017). The unified framework for biological
1512 invasions: a forest fungal pathogen perspective. *Biological Invasions*. doi: 10.1007/s10530-
1513 017-1450-0.

Wingfield, M.J., and Swart, W.J. (1994). Integrated management of forest tree diseases in South-Africa *Forest Ecology and Management* 65(1), 11-16. doi: 10.1016/0378-1127(94)90253-4.

Woodward, S., Stenlid, J., Karjalainen, R., and Huttermann, A. (1998). *Heterobasidion annosum Biology, Ecology, Impact and Control.* Wallingford: CAB International.

Zeglen, S., Pronos, J., and Merler, H. (2010). Silvicultural management of white pines in western North America. *Forest Pathology* 40(3-4), 347-368. doi: 10.1111/j.1439-0329.2010.00662.x.

9 Author Contributions

JRH conceived the study. AK, NB, CG and JH acquired funding for the project. JH and MR designed the study. MR carried out data collection, performed the analysis and led the drafting of the manuscript, with input from JH. All authors discussed and interpreted the results and contributed to the writing of the final manuscript.

10 Funding

This work is from the project titled *Modelling economic impact and strategies to increase resilience against tree disease outbreaks*. This is one of seven projects in the Tree Health and Plant Biosecurity Initiative (phase 2) funded by BBSRC, Defra, ESRC, Forestry Commission, NERC and Scottish Government. The Rural & Environment Science & Analytical Services Division of the Scottish Government provided supporting capacity to MR for final editing of the paper.

11 Acknowledgments

The authors thank the other members of the FOREMOD project team (Chris Quine, Morag Macpherson, Ciara Dangerfield and Oleg Sheremet) for valuable discussions and insights with have contributed to the development of this paper in many ways.

12 Conflict of Interest Statement

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

13 Data Availability

All datasets generated for this study are included in the manuscript and the supplementary files.

1549 **Table 1. Overview of coverage of identified papers**

1550

Location	Percentage of papers
North America (excluding California and Oregon coastal forests)	35
California and Oregon coastal forests	24
Europe	29
Other	11
Tree type	
Broad leaves	48
Conifer	52
Pathogen type	
Fungi (excluding Armillaria)	48
Armillaria	17
Oomycete (all phytophthora)	21
General	14

1551

1552

1553

1554 **Table 2. Overview of published evidence of effects of forest management on resilience to**
1555 **tree diseases.**

1556

Identification and management of sources of primary infection

Management	Disease indication	Trend	References
Connectivity	Mortality	Increased	(Jules et al., 2002)
	Disease incidence	Increased	(Hessburg, 2001; Condeso, 2007; Cushman and Meentemeyer, 2008; Meentemeyer et al., 2008a; Ellis et al., 2010; Haas, 2011; Goheen et al., 2012; Peterson et al., 2014)
	Disease severity	Increased	(Condeso, 2007; Haas, 2011; Havdova, 2017) (Meentemeyer et al., 2008a; Meentemeyer et al., 2008b)
	Recovery	No data	
Previous land use	Mortality	No data	
	Disease incidence	Varied	(Puddu et al., 2003; Meentemeyer et al., 2008a)
	Disease severity	Increased in previous forest land	(Meentemeyer et al., 2008a)
	Recovery	No data	
Site preparation	Mortality	Decreased	(Morrison et al., 1988; Gibbs, 2002; Hood et al., 2002; Thies and Westlind, 2005; Whitney and Irwin, 2005; Cram, 2010; Richter et al., 2011b; Shaw et al., 2012; Cleary et al., 2013; Morrison et al., 2014)
	Disease incidence	Varied	(Morrison et al., 1988; Ronnberg, 2000; Hood et al., 2002; Pankuch et al., 2003; Thies and Westlind, 2005; Thor and Stenlid, 2005; Holzmueller et al., 2008; Hood and Kimberley, 2009; Cram, 2010; Fichtner et al., 2011; Kwon

			et al., 2011; Richter et al., 2011a; Beh et al., 2012; Shaw et al., 2012; Cleary et al., 2013; Valachovic et al., 2013b; Bauman et al., 2014; Crone et al., 2014; Morrison et al., 2014; Roach et al., 2015)
	Disease severity	Varied	(Blodgett et al., 1997; Filip et al., 2002; Anglberger and Halmschlager, 2003; Thies et al., 2006; Halmschlager and Katzensteiner, 2017)
	Recovery	Varied	(Thies and Westlind, 2005; Begley et al., 2009; Morrison et al., 2014)

Management of sources of secondary infection

Management	Disease indication	Trend	References
Tree establishment under alternative silvicultural systems	Mortality	Varied	(Ostry, 2000; Katovich et al., 2004; Ferchaw et al., 2013)
	Disease incidence	Varied	(Ostry, 2000; Katovich et al., 2004; Filip et al., 2010; Ward et al., 2010; Ostry et al., 2012; Ferchaw et al., 2013)
	Disease severity	Decreased	(Rosenvald et al., 2015)
	Recovery	Varied	(Filip et al., 2010; Ferchaw et al., 2013)
Canopy cover	Mortality	No data	
	Disease incidence	Varied	(Campbell, 2000; Ellis et al., 2010)
	Disease severity	Increased	(Condeso, 2007)
	Recovery	No data	
Tree density	Mortality	Increased	(Burdon et al., 1992; Gerlach et al., 1997; Bishaw et al., 2003)

	Disease incidence	Increased	(McCracken and Dawson, 1998; Bergdahl et al., 2002; Bishaw et al., 2003; Piirto and Valkonen, 2005; Staudhammer et al., 2009; Dillon et al., 2014)
	Disease severity	Increased	(DesprezLoustau and Wagner, 1997; McCracken and Dawson, 1998; Mattila et al., 2001; Mattila, 2002; Meentemeyer et al., 2008a; Dillon et al., 2014; Havdova, 2017)
	Recovery	No impact	(Bishaw et al., 2003)
Thinning	Mortality	Varied	(van der Kamp, 1994; Robinson, 2003; Baleshta et al., 2005)
	Disease incidence	Varied	(Hungerford et al., 1982; Harrington et al., 1983; Entry, 1991; van der Kamp, 1994; Rosso and Hansen, 1998; Nebeker et al., 1999; Hessburg, 2001; Morrison et al., 2001; Hood et al., 2002; Kaitera, 2002; Pankuch et al., 2003; Robinson, 2003; Thor and Stenlid, 2005; Leak, 2006; Dwyer et al., 2007; Otrosina et al., 2007; Bulman, 2008; Hood and Kimberley, 2009; Oliva et al., 2009; Meadows et al., 2013; Roach et al., 2015)
	Disease severity	Varied	(Morrison et al., 2001; Roach et al., 2015; de Urbina et al., 2017)
	Recovery	Increased	(Amorini et al., 2001; Morrison et al., 2001; Hood and Kimberley, 2009; Meadows et al., 2013)
Diseased tree removal	Mortality	No data	
	Disease incidence	Decreased	(Kanaskie et al., 2006; Ganley and Bulman, 2016; Menkis et al., 2016)
	Disease severity	Decreased	(Amorini et al., 2001; Greene et al., 2008)
	Recovery	No data	
	Mortality	Decreased	(Lavallee, 1991)

Pruning and coppicing	Disease incidence	Varied	(Hungerford et al., 1982; Chou, 1988; Hagle, 1988; Lavallee, 1991; Madar et al., 1991; Metzler, 1997; Ostry, 2000; Hood et al., 2002; McCracken and Dawson, 2003; Pleysier et al., 2006; Tarigan et al., 2011; Valachovic et al., 2013a; Sakamoto et al., 2016)
	Disease severity	Varied	(Ostry, 2000; Martin et al., 2005; Bezos et al., 2012; Sakamoto et al., 2016; de Urbina et al., 2017)
	Recovery	No data	
Species diversity and mixtures	Mortality	Decreased (dependant on species mixture)	(Heybroek, 1982; Gerlach et al., 1997; McCracken and Dawson, 1997; 1998; D'Souza et al., 2004; Simard et al., 2005; Ramage et al., 2012; Morrison et al., 2014)
	Disease incidence	Decreased (dependant on species mixture)	(Benedict, 1981; Gerlach et al., 1997; McCracken and Dawson, 1997; Campbell, 2000; Mattila et al., 2001; Peacock et al., 2001; Bergdahl et al., 2002; Linden and Vollbrecht, 2002; Mattila, 2002; Puddu et al., 2003; Santini et al., 2008; Begley et al., 2009; Oliva et al., 2009; Davidson et al., 2011; Haas, 2011; Hantsch, 2013; Haas et al., 2016; Nguyen et al., 2016)
	Disease severity	Decreased (dependant on species mixture)	(Hantsch, 2013; Hantsch et al., 2014a; Hantsch et al., 2014b; Havdova, 2017)
	Recovery	Increased (dependant on species mixture)	(Gerlach et al., 1997; McCracken et al., 2001; Baleshta et al., 2005; Simard et al., 2005; Begley et al., 2009; Morrison et al., 2014)

1557

1558

1559 **Table 3. Summary of studies in tree species diversity/mixture effects.**

1560

Pathogen	Tree species	Location	Diversity measure	Planted tree species mixture considered ?	Effect	Reference
General disease	General forest	Europe (multiple locations)	Forest tree diversity	No	Reduced disease incidence	(Nguyen et al., 2017)
Fungal infections	Broadleaf	Germany	Species richness	No	Reduced disease incidence in most susceptible species	(Hantsch et al., 2014a)
Fusarium sp. canker	Sugar Maple	Canada	Tree diversity	No	Reduced disease incidence	(Bergdahl et al., 2002)
P. ramorum	Oak forest	California	Species richness	No	Reduced disease incidence	(Haas, 2011; Haas et al., 2016)
P. ramorum	Douglas fir	California	Tree diversity	No	Reduced mortality	(Ramage et al., 2012)
Armillaria sp.	Silver fir	Spain and Italy	Pure vs mixed stands	No	Increased isolation of pathogen from soil in mixed stands	(Oliva et al., 2009)
Root rot	Lodgepole pine	British Columbia, Canada	Mixtures containing cedar and birch	Yes	Reduced mortality	(Morrison et al., 2014)

Root rot	Lodgepole pine	British Columbia, Canada	Mixtures containing Douglas fir	Yes	Increased mortality	(Morrison et al., 2014)
P. cinnamomi	Eucalyptus	Australia	Mixture containing *Acacia pulchella*	Yes	Reduced mortality (not seen with other *Acacia* species)	(D'Souza et al., 2004)
H. annosum	Norway spruce	Norway	Mixture containing Scots pine	Yes	Reduced disease incidence	(Linden and Vollbrecht, 2002)
Fungal infection	*Tilia cordata*	Germany	Mixture containing Scots pine	Yes	Reduced disease incidence	(Hantsch et al., 2014a)
Fungal infection	*Quercus petraea*	Germany	Mixture containing Norway pine	Yes	Increased pathogen load	(Hantsch et al., 2014a)
Fungal infection	*Tilia cordata*	Germany	Mixture containing European beech	Yes	Increased pathogen load	(Hantsch et al., 2014a)
Ash dieback	Ash	Czech Republic	Mixture containing *Abies* and *Acer* species	Yes	Reduced disease severity at stand level	(Havdova, 2017)
Ash dieback	Ash	Czech Republic	Mixture containing oak species	Yes	Increased disease severity at stand level	(Havdova, 2017)

Armillaria root rot	Douglas fir	USA	Mixture containing conifers	Yes	Increased mortality	(Gerlach et al., 1997)
Armillaria root rot	Douglas fir	British Columbia, Canada	Mixture containing conifers	Yes	Increased mortality	(Baleshta et al., 2005; Simard et al., 2005)
Melampsora pinitorqua	Scots pine	Finland	Mixture containing aspen and willow	Yes	Increased disease incidence	(Mattila et al., 2001)
Melampsora pinitorqua	Scots pine	Finland	Mixture containing aspen	Yes	Increased mortality	(Mattila, 2002)
Fungal infection	Mixed forest	Germany	Mixture containing *Qurecus petraea*	Yes	Increased pathogen load	(Hantsch, 2013)
Melampsora epitea	Willow	Ireland	Clone diversity	Yes	Reduced mortality for most susceptible clones	(McCracken and Dawson, 1998)
Melampsora epitea	Willow	Ireland	Clone diversity	Yes	Later disease onset within stands under 4 years old	(McCracken and Dawson, 1997)
Melampsora epitea	Willow	Northern Ireland	Clone diversity	Yes	Increased growth rate	(McCracken et al., 2001)

Melampsora epitea	Willow	Northern Ireland	Clone diversity	Yes	No change in mortality, but higher growth in the first 3 year harvest cycle.	(Begley et al., 2009)

1561